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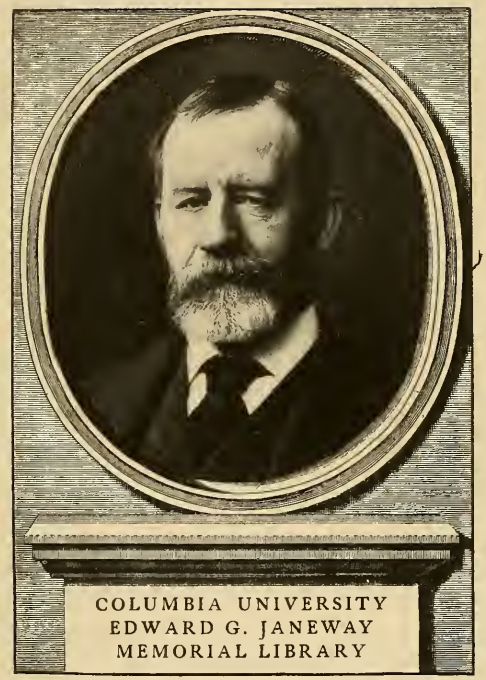

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THE CAUSES AND PREVENTION OF PHTHISIS

THE MILROY LECTURES

1890

The Milroy Lectures for 1890

THE

CAUSES AND PREVENTION

OF

PHTHISIS

BY

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LONDON
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1890

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PREFACE

The subject of the Milroy Lectures for the year 1890 was selected, notwithstanding its apparent triteness, and the numberless treatises that have been written upon it, partly for its own intrinsic importance, but mainly because it is one to which Dr. Milroy himself gave much prominence.

He mentions it again and again in his suggestions; 1st, in regard to the 'cachexias' that may constitute a pre-tuberculous stage of the malady (p. 1); 2ndly, directly, under the head of tuberculosis (p. 2); 3rdly, in the suggestion that it is 'based upon, and connected or associated with an unhealthy condition of the constitution, or habit of the system. This unhealthy condition, or dyscrasia of the body, is often,' he says (p. 2), 'doubtless, in respect of some of these maladies, congenital and hereditary; but observation seems distinctly to show that it may also be acquired and become developed *de novo*, independently of parental transmission or descent; and that in such cases the genesis or origination of the malady will probably be found to be largely due to the neglect of some of those natural laws of healthy existence, with the consideration of which the Science of Public Hygiene professes to deal.'

Fourthly, in his comparison of these cachexias with leprosy he remarks: 'If not the direct offspring of appreciable unwholesome and insanitary influences or local conditions, there is certainly reason to believe that the continuance, extension, and inveteracy of the

maladies are under the control of judicious hygienic precautions, and that, towards their diminution, and possibly their eventual extinction, much may be done by—

(*a*) The amelioration of the food of the working classes;

(*b*) The internal improvement of their dwellings;

(*c*) The amendment of their habits as to personal and domestic cleanliness, temperance, &c;

(*d*) Coupled with the drainage of malarial lands near to their habitations, and the selection of healthy sites for these in the first instance.

Fifthly, he speaks of the 'propagation of tubercular disease, and especially of tubercular consumption, by contagious transmissibility.'

And lastly, though incidentally, he adverts to the necessity 'of more diligent and continued attention to the study of the accurate geography of diseases, together with the exact chronology of the appearance and persistence of those diseases which are of only occasional and temporary occurrence, and also the exact date of each epidemic prevalence, or extra severity of the ordinary *endemic* maladies in different countries and localities.'

In the following lectures, each of these suggestions is considered in its turn, and an attempt is made to bring the subject in line with the most recent researches respecting the causes of tuberculosis, and the modes in which it may most readily be prevented.

Some slight changes in the exact arrangement of the lectures have been made, but they are now published in form and substance practically as they were delivered.

1 St. Peter's Square, Manchester,
April 1890.

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THE

CAUSES AND PREVENTION OF PHTHISIS

LECTURE I

PATHOLOGY AND AFFINITIES OF TUBERCLE

The mortality from phthisis—Its curability and preventibility—Search for infective material—Koch's discovery—Pathology of tubercle—Toxic principles evolved by bacillus—Conditions of growth—Course of infection—Relation to other pathological organisms—Contrast with epidemic disorders—Affinities with Typhoid Fever—Ague—Glanders—Actinomycosis—Frambœsia — Madura-foot — Syphilis — Cancer—Leprosy—Close relations with leprosy—Micro-organism—Pathology—Endemicity—Geographical distribution — Contagion — Heredity—Question of identity—Practical conclusions.

Tubercle, in its various forms, at the present day, carries off annually nearly 70,000 persons.

In the form of phthisis, at ages between 15 and 45—the most useful stages of human existence—it kills more than one-third of the people who die, and nearly half between 15 and 35.

Moreover, in its prolonged and painful course, it either prevents its victims from earning their livelihood, or at least interferes greatly with their daily work. Its habit of seizing upon the flower of the population; its slow, but, in the majority of cases, almost certain, progress towards death; the distressing weakness and suf-

fering of the last few weeks or months of existence; all these are features in the fell disorder that render its study all-important, not only to medical men, but also to statesmen, and to all who are concerned with the welfare of the nation.

It is, perhaps, hardly necessary before my present audience to defend the thesis, that phthisis is both curable and preventible. A great change has of late years been wrought in medical opinion upon this subject. Up to quite a recent period, not only was consumption supposed to be incurable, it was also regarded as almost inevitable. Families in which existed a taint of the disease were supposed to be doomed to lose some of their members from this cause. Insurance offices still refuse to enrol upon their books those who have lost a father and mother from the disease; and even collateral relatives, who have died from it, are judged to have an influence upon the candidate for assurance.

The fate of the consumptive himself was also generally regarded as hopeless. It must be in the memory of most of those now present to have seen the recognition of even the early stages of tubercular disease regarded as equivalent to a sentence of death, and the dictum of Sir Thomas Watson, that 'tubercular disease, when established, is beyond our power,' [1] was currently accepted as the truth.

These views of the inevitable character and the incurability of phthisis are now altered. Thus Niemeyer remarks [2]: 'Many a patient gets well who would formerly have been assumed to be the victim of tubercular, and therefore incurable, disease.' Dr. Pollock shows [3] that many cases which were given up by doctors, but outlived

[1] *Practice of Physic*, ii. 201. [2] *Lectures on Consumption*, p. 65.
[3] *Elements of Prognosis in Consumption*, p. 68.

the prediction to arrive at old age, were undoubtedly recoveries from phthisis. Many more were instances of an early invasion of the disease, with subsidence of, and long toleration of, the deposit;' and again (p. 17), 'the best authorities lean to the opinion that tubercle is capable of removal by absorption.'

In his work on Pulmonary Consumption (p. 324), Dr. C. T. Williams gives, as the result observed in private practice, that 'a cure was effected in 4·6 per cent. of the cases; great improvement in 38 per cent.; the disease was stationary in 13·4 per cent.; but in 43·5 per cent. there was more or less increase.'

I have myself a list of more than 40 persons, who have been under my own observation for many years, in whom the disease has made no progress and who may therefore be considered as practically cured.

The evidence derived from post-mortem examinations of adults who have died of other diseases than phthisis, also shows that a very large percentage of persons have suffered and have recovered from tubercular disease of the lungs.

My colleague, Dr. Harris, has himself investigated this subject, and found that in at least 39 per cent. of the autopsies at the Manchester Royal Infirmary, there was evidence of cured phthisis. He also refers (in a paper on the curability of phthisis) [1] to the researches on this point of Cruveilhier, Rogée, and Dejerini, 50 per cent. of their cases having shown signs of caseous and calcareous formations. Bollinger, in 256 similar cases, found 27 per cent. with evidence of healing; Standacker, 26 per cent., and Marsini 39 per cent. Evidently a very large proportion of our population suffers from tubercular disease and recovers from it.

[1] *British Medical Journal*, Dec. 21, 1889.

With regard to the *preventibility* of the disease, still less can any doubt prevail. In the last 30 years, if we take the returns of the Registrar-General as our authority, the annual rate of mortality from phthisis has been reduced by more than one-third. In the three years 1858–60, the rate per million of persons living was 2,567, in 1888 it was only 1,541, a diminution of more than 1,000 per million, and, if we take the population as over 29,000,000, this means a saving of about 30,000 lives every year, from this cause alone.

Nor is this improvement confined to England. 'In 1857, 39·50 deaths from consumption were returned in the State of Massachusetts for each 10,000 of the population; in 1883, only 29·90. This decrease is too large to credit to greater accuracy in diagnosis and to the transference of consumption to other States, and is mainly attributable to the prevention of phthisis by improved hygiene.' [1]

Evidence has been also forthcoming, of the strongest kind, of the influence of sanitary measures, and especially of good drainage and good ventilation, as preventive of consumption.

No better instance of this could be found than in the records of the mortality from the disease in the British army and navy. This evidence was collected by the Commission on the sanitary state of the army, 1858, and the results are shown in the following table, which has frequently been quoted before, but which can hardly be too often brought before public notice. This mortality is given at several stations for several successive periods,

[1] *Strumpel Text-book of Medicine*, p. 213. It is interesting to note that in Canada no similar reduction has taken place in the phthisis-rate. In eleven years, 1870 to 1880, the rate per 1,000 deaths was 11·4, and in ten years, 1878 to 1887, it was 12·4.

and I think that you will be at once struck by the enormous death-rates amongst the troops in the earlier periods and at every one of the stations, and the great reduction of the rate in 1874. At the present time it is still further reduced. In 1883, it was only 6·28 per 1,000, and throughout the world only 9·57.

TABLE I.—*Mortality per* 1,000 *of strength.*

	1830 to 1837.	1837 to 1847.	1863 to 1872.	1874.
Household Cavalry	14·5	11·1	9·17	8·79
Cavalry of Line	15·3	13·5		
Foot Guards	21·6	20·4		
Mediterranean Stations	21	16·4	11·2	7·27
Canada, &c.	23	17	9·49	6·0
Jamaica, &c.	91	59	17·05	16·9
Madras, India	52	—	24·2	14·22
Bengal ,,	44			
Ceylon	49	—	21·95	6·04

Rates of Mortality at the same ages prevailing in healthy country populations	7·7
In England and Wales	9·2
In Manchester	12·4

The greater part of this excessive mortality was due to consumption. Dr. Buchanan, now medical officer to the Local Government Board, has also shown conclusively that good drainage of a locality may diminish by one-half the prevalence of the disease, as in the case of the city of Salisbury.

These instances may suffice to show that we are entering upon no barren quest in searching for means, to reduce still further the mortality from consumption.

So much has already been said upon the subject, that it might well seem somewhat presumptuous in me to attempt to re-tread the path that has already been opened up, and so well explored, by men like the two

Williamses, Southey, Ancell, Treves, and a host of others.

Every organ and every structure of the tuberculous subject has been carefully examined, weighed, and measured. Their tissues have been searched for abnormal signs, and analysed to discover alterations in their chemical composition. The blood and all the fluids have likewise been scrutinised by the aid of the microscope, and subjected to chemical research. The functions of the living body have been watched in all their phases—in brief, all the forces of the animal economy have been reckoned up, and the special pathology of tubercle thoroughly searched out.

But medical science does not stand still, and since most of the work thus alluded to was performed, great additions have been made to our knowledge of the subject.

Koch's great discovery alone would have rendered it desirable, and even necessary, to pass under review the older researches into the etiology and prophylactic treatment of consumption.

It is now eight years since Professor Koch succeeded in demonstrating the existence of a specific organism in all tubercular diseases. By pure cultivations of the bacillus, and by numerous experiments upon animals, he may be said to have fully proved its essential relation to the disease.

But, notwithstanding the confidence inspired by the character and methods of the discoverer, some little time was needed to establish the truth, and to obtain confirmation of his observations by others.

The early years after a great discovery are often thus taken up with its verification and its application to previously ascertained facts. The master builder has laid a head corner-stone, and other minor workers are

busily engaged in raising other portions of the edifice to its level.

Such an epoch is now before us, and the full effect of Koch's great discovery, upon our estimate of the secondary causes of phthisis, has even yet hardly been correctly ascertained.

The earlier years after 1882 were, in fact, mainly occupied with the researches of rival pathologists. Aufrecht, Spina, Klebs, Schottelius, Eberth, Malassez, and Pignal, and many others, discussed the essential influence of the newly-found organism, and the possibility of zooglœa-like forms of tubercle, or of pseudo-tubercle.

Clinical researches were undertaken in all countries, notably in England by Watson-Cheyne, Dreschfeld, and C. T. Williams; in France by Chantemesse, Duguet, Gosselin, Héricourt, and Warlomont; in Italy by Celli, Cavagnis, Guarnieri, and Voltolini; in Germany, by Baumgarten, Biedert, Langerhans, Veraguth, and a host of others.

Numerous experiments on the inoculation of tubercle-bacilli were undertaken, and researches were made as to the presence of the virus in milk and other foods, in the air, in dust, in walls, and even in house-flies. Some of these will have to be reviewed later on. On the whole, it may be said that Koch's position has been only more firmly established by these observations, and that it must be acknowledged that, whatever may be its mode of working, the tubercle-bacillus is the specific cause of the disease.

I can hardly hope to add many stones to the still unfinished work; but at least it may be of some advantage, to attempt to bring into one field of view the various facts bearing upon the natural history of the organism,

its favourite haunts and breeding-grounds, the soil most favourable to its growth, the food that will nourish it into virulence, and, most important of all, the conditions under which it may be robbed of its power for evil.

One consequence of the successful search for the *vera causa* of tubercle, and that perhaps not the least important, is, that it thrusts aside all former speculations as to the nature of tuberculous growths.

There is now no need of learned and elaborate inquiries as to whether tubercle is 'a blastema, coagulating near the vessels from which it exudes,'[1] or 'a deposit of specific tubercle cells,'[2] or 'a retrograde metamorphosis of tissues,'[3] or 'a lymph-tumour, a new-growth arising out of connective tissue by endogenous multiplication,'[4] or 'a retention in the blood of degenerate substances,'[5] or 'lymph, malformed, oxidised, and precipitated from the blood upon contact with air.'[6] All these surmises are now of interest mainly as matters of history. Scrofula and phthisis have also been shown to be due to one and the same cause. Lupus is recognised as tubercular disease of the skin; and the various forms of tubercle itself are seen to be due, primarily, to the extent of the irritation caused by the presence of the parasite and its products; secondly, to the efforts made by the surrounding parts to resist the invader.

The essential pathology of phthisis may, in fact, be stated very briefly. Dr. C. T. Williams says: 'Reaching the alveolus through the breath, the bacillus enters the epithelial cell and causes proliferation by irritation. The alveolus becomes stuffed with cells, and if the irritation be a gradual process, it gives rise to a highly nucleated product in the giant-cell. If the irritation be great,

[1] Rokitansky. [2] Lebert. [3] Addison.
[4] Virchow. [5] Buhl. [6] Simon.

caseation is rapidly produced, though whether this be due to necrobiosis from overcrowded proliferation, or, as Dr. Watson Cheyne thinks, to a chemical change in the cells, brought about by the action of the bacilli, is not yet determined.'[1]

Dr. Watson Cheyne himself says, 'Bacilli are inhaled into the air-cells of a lung which is in a fit state, whether by predisposition or otherwise, for their growth. They at once attack the epithelial cells, and, in the first instance, cause their hypertrophy and multiplication. The bacilli growing in these cells produce poisonous chemical substances, and the cell which in the first instance was stimulated to increased growth by a small quantity of the poison, soon succumbs to the increasing amount and undergoes caseation. Some cells, or groups of cells, are, however, stronger than the others, and go on growing so as to form giant-cells. These generally ultimately succumb, though in some instances they may get the upper hand, and the bacilli may disappear. While this is going on inflammation spreads around, and the process crosses from air-cell to air-cell. I believe also that this view of the production of a poisonous substance by the bacilli may explain the fever and wasting of phthisis.'[2]

These observations are also confirmed by others. For instance, Veraguth,[3] as a result of his experiments on the inhalation of tuberculous sputum, notes that the first change to be seen is an increase in the number of tubercle-bacilli in the alveolar epithelium, with consequent desquamation; then, later on, hyperæmia with migration of white blood-corpuscles into the alveoli, and

[1] *Pulmonary Consumption*, 2nd ed. p. 44.
[2] *British Medical Journal*, 1885, i. p. 170.
[3] *Arch. f. Exper. Path.* Band xvii.

a reactive increase in the neighbourhood of the disease-focus, which tries thus to keep off the bacillus by encapsuling itself.

Through the presence of the bacilli caseation takes place, the central portion of which dies. Only, on the periphery of the diseased focus, within epithelioid or giant-cells, certain tubercle-bacilli or spores are preserved, which later on enter the neighbouring lymph-tracks, and set up fresh foci in the interstitial connective tissues and in the lymph-glands.

Baumgarten has even watched the development of epithelioid and giant cells,[1] and describes the so-called epithelioid cells as arising from the action of the tubercle-bacilli on the fixed cells of the connective tissues, or on the epithelium. 'Out of the epithelioid cells,' he says, 'the giant cells proceed. An increase of the nuclei takes place in them, while the partition of the cell-body is omitted. This happens the more frequently the fewer there are of the tubercle-bacilli, and the weaker their power of growth. If, however, an abundance of these bacilli are present, or their power of growth is increased, an irritation is caused in the walls of the vessels, and the white corpuscles migrate, and gradually supplant the epithelioid cells, thus taking the upper hand in building up the disease already commenced.'[2]

Weigert[3] also confirms this story of the formation of the giant-cells, by noting that there is a partial proliferation of the cell-nucleus, whilst the body of the cell does not divide; then, in consequence of a partial caseation of the cells, the protoplasm is held together by the

[1] *Zeitschrift f. klin. Mediz.*, Band ix. & x.

[2] See also Cornil, *Etudes Expérimentales et Cliniques sur la Tuberculose*, Verneuil, Paris, 1887.

[3] *Deutsche med. Wochenschrift*, 1885, No. 35.

dead, and apparently degenerated, portions of the growth. Hence, the tubercle-bacilli lie, not in the centre of the cell, but in the neighbourhood of its nucleus, because, through the caseation of the protoplasm they go to the ground, but in the proliferating zone they can sustain and increase themselves.

It is thus plain that Dr. Williams' account of the relationship of the bacillus to the pathological changes in tubercle is fully borne out by these observers, and that a clear and intelligible histology of the disease has been worked out. In Ziegler's words, we must 'regard tubercle as an inflammatory product, the result of an irritation, an infection;'[1] and, as we have seen, such constant association of the bacillus with the lesions of phthisis by no means excludes the possibility, often insisted on, of the secretion or production by its means of certain unorganised morbific material.

Dr. Watson Cheyne attributes much importance to this secretion, and Dr. Wilson[2] says, 'the constitutional manifestations of tuberculosis are not directly due to the bacilli, but to toxic principles evolved during their growth and multiplication.' He quotes Nencke's chemical analysis of tubercle-bacilli, and his separation from them of a tetanising poison; also Bonardi's demonstration in tuberculous sputum of certain organic bases which caused disturbance of the nervous system when introduced into rats and guinea-pigs.

Dr. Coats, in his 'Lectures to Practitioners' (p. 185), seems also inclined to infer that it is not the bacilli themselves, but their products, that are harmful. 'They are minute vegetable bodies,' he says, 'which multiply

[1] *Tuberculose und Schwindsucht*, p. 279.

[2] 'Etiology and Prophylaxis of Tuberculous Diseases,' *Medical News*, June 8, 1889.

along surfaces or channels, and do not usually penetrate into the living tissues. Such minute bodies cannot, by their merely physical properties, produce any serious damage. It must be rather that, in their growth, they evolve certain chemical principles which are injurious to the tissues.' He adduces, in proof of his contention, the fact that, in tuberculosis of the peritoneum, you have the flat yellow caseous tubercles buried in new-formed connective tissue, which is fully organised, and contains blood-vessels; 'it is the diluted products of the bacilli that induce the more simple inflammatory lesions.' Pleuritic infection in phthisis he considers to be due to the same cause, and that a good deal of the inflammatory lesion in the connective tissue of the lung itself is of a similar nature.

These brief remarks may perhaps suffice as a statement of modern views respecting the pathological histology of tubercle.

It is also unnecessary, before my present audience, to recapitulate at length the characteristic points connected with the development and growth of the bacillus. They were so fully made out by Professor Koch himself that scarcely any addition has been made to our knowledge in this direction. For our present purpose, it will be sufficient to recall to our minds (1) the small range of temperature within which the organism can be successfully cultivated (between 84° and 107° Fahr.); (2) the length of time, from two to three weeks, required for its development; (3) its tenacity of life in sputum, Fischer and Schill having found it virulent after 43 days in putrefying sputum, and 186 days in sputum dried at ordinary temperatures; (4) the observation showing the antagonism that exists between the living elements, especially the wandering cells, the leucocytes of the body, and the

bacillus—observations tending to show that, as a rule, the organism has a short life within the body, and that it can only maintain its presence, for any length of time, by the appearance of new generations to fill the places of the dying.[1]

The histology of tubercle being thus clear, the reason for its spreading infectively through the system has also been made plain. It hardly needed Koch's demonstration of the bacillus, to show the track by which it usually makes its way. A close relationship between the lymphatic system and tubercle had already been clearly shown by many observers, such as Virchow, Wedl, Rindfleisch, Wagner, Aufrecht, Buhl, Burdon-Sanderson, and Treves.

Thus Rindfleisch[2] pointed out that 'specific irritation of the endothelia of the lymphatics, the serous membranes, and the blood-vessels, is the essential factor in the production of the miliary nodule, and it is only because the lymphatics run by preference in the immediate neighbourhood, in the adventitia of the blood-vessels, that miliary tubercles exhibit a preference for that locality.'

Aufrecht regarded miliary tubercle as 'a granular perilymphangitis.'

Wagner speaks of 'the extension of tubercle . . . in the course of lymphatic vessels, or in that of blood-vessels.'

Treves looks upon tubercle as infective, and affirms that 'lymphatic structures of some kind are essential' to its formation.

Buhl[3] goes still further and says, 'Tubercle extends from the internal wall of the alveoli into the textureless lymphatic vessels.'

These ante-bacillary observations are only confirmed

[1] Koch, *Etiology of Tuberculosis*, Syd. Soc., i. 98.
[2] *Path. Histology*, Syd. Soc., i. 141. [3] *Lungenentz*, p. 106.

by the researches of Koch, and by such men as Baumgarten, Fischer, Volkmann, and others.

Also the mode in which the bacillus gains access to the system was noted long before Koch's time. It was recognised that the points of attack selected by the disease indicated that the infection comes from without the body, thus Cohnheim, in his work 'Die Tuberculose vom Standpunkt der Infectionslehre' (p. 20), shows that a tuberculous or scrofulous product is mostly deposited in those parts of the body that are either most exposed to attack from without, or in which any virus coming from the outside may lodge for the longest time. Thus no internal organs are so constantly brought into relation with the atmosphere as the lungs, and accordingly no other organ is attacked by tubercle with the same frequency and the same intensity.

In many cases of this disease also all the other organs, not connected with the lungs, are free from its attack.

Such a fact as this could not well be accounted for on any other supposition than that of a primary and immediate attack upon the respiratory tract by the virus. Next in order to the lungs and pleuræ, and to the bronchial and tracheal glands, come the glands about the pharynx; and then, next after these, owing probably to the swallowing of sputa, we find the lymphatic apparatus of the walls of the intestines, the isolated and agminated follicles, most liable to be the seat of tuberculous ulceration. The œsophagus escapes contagion because of the rapidity of the transit of the virus, and the stomach, perhaps, owing to the presence of the gastric juice. Through the intestinal ulcers a path is opened to the mesenteric glands, the liver, and the whole of the rest of the body, thereby producing general tuberculosis.

The demonstration by Koch of an organism that must in most cases come from the air, and that could in this way penetrate the lungs, or enter the lymphatics by sores, only served to make assurance doubly sure. The vehicles of the virus were indicated. Tubercular bacilli were found by Koch in dried and powdered sputum, by Cornil in the walls of rooms, by Dr. C. T. Williams in the air brought by ventilating-shafts from a ward in Brompton Hospital, and by myself, though rarely, in the aqueous vapour condensed from the breath of patients in an advanced stage of consumption, and this latter observation was confirmed by Dr. C. Smith by means of gun-cotton respirators.[1] The point of usual entrance of the organism was also demonstrated.

Professor Klein followed the development of tubercle from the 'epithelial cells of the alveoli,' and, as we have seen, Dr. Watson Cheyne confirmed these observations by finding that the principal seat of the specific bacillus in

[1] Some doubt has been cast upon the possibility of bacilli being found in the breath, especially by Dr. Cornet (*Berliner Mediz. Woch.*, 25 März, 1889, p. 250). He adduces as grounds for disbelief the considerations, 1. that non-volatile substances could not be exhaled; 2. that many competent observers have failed to find them; 3. that errors may have crept into the observations. To these objections I would reply: 1. That anyone who will examine microscopically the condensed aqueous vapour of the breath may soon convince himself that it contains much non-diffusible organic matter, such as epithelial scales, &c. (see paper on 'The Organic Matter of the Breath,' *Jour. of Anat. and Phys.*, iv. 209). 2. To the observations named by Dr. Cornet must be added those of Dr. G. Heron, who informs me that he also was unable to find bacilli in tubes and watch-glasses exposed for long periods to the breath of phthisical patients. But there is nothing surprising in the fact that such negative results have been obtained. I found no bacilli, in some cases, in the deposit from several drachms of condensed aqueous vapour, and very few in any case. 3. The fact that the bacilli were so few in number is of itself a tolerably sure proof that no contamination by sputum had taken place, owing to the precautions taken to prevent this accident.

the lungs is the alveolar epithelium, and its products, *i.e.* in the epithelioid cell and the giant-cell.

Another important result of Koch's discovery is that in placing tuberculosis undoubtedly amongst the class of microbic diseases, it imposes upon us the task of determining its affinities and assigning to it its place amongst other such disorders.

It was suspected long ago that tubercle bore a close relationship to some of the infective class of diseases.

Without laying any stress upon the ancient guess that consumption is due 'to animalcules in the lungs,'[1] it was proved by Villemin in 1865 that tuberculous material would convey the disease to sound animals, when it was introduced into their bodies by puncture or inoculation, and his results were shortly afterwards confirmed by others. Moreover animals fed with this material contracted the complaint, and the inhalation of dried and pulverised sputum from consumptive patients caused the disease in dogs and other animals.

Many observers were also on the look-out for specific organisms, notably Chauveau, Baumgarten, Ziegler, and Zinn. Ziegler[2] plainly tells us to regard tubercle as 'an inflammatory product, the result of an irritation, an infection,' and he does not doubt that 'schizomycetes play an important part in phthisis.'

Some, as Klebs and Deutschmann, thought they had actually found a 'monas tuberculosis;' Toussaint a 'coccus;' Buhl and Aufrecht, 'a specific bacterium.'

Finally Koch's researches settled the question, and the tubercle-bacillus reigns supreme as the essential specific element in the causation of phthisis and other tubercular diseases.

[1] Martin, 1722.

[2] *Tuberculose und Schwindsucht*, p. 279.

But even before the specific cause was established, the relationship of tubercle with other infective disorders had been suspected.

Both Villemin and Virchow compared tubercle to the granulations of glanders, and the gummata of syphilis; and though the former separated scrofula from tubercle, he points out that they both share with syphilis, glanders, and typhoid fever, the character of forming caseous material. Klebs and Cohnheim also pointed out the similarity existing between tubercle, glanders, syphilis, lupus, pearl disease in cattle, and even lepra, and they regard them as closely allied in all essential points of their anatomical structure and history.

In his Gulstonian Lectures on Tubercle, Dr. Southey also showed that typhoid lymphomata are very like tubercle, both in their composition and their course, affecting similar parts and leading to like lesions. He places tubercle between cancer and these and other lymphomata.

Dr. Southey goes so far as to suggest that tubercle in its acute form may even become epidemic, and he cites, as instances of this form, some cases recorded by Dr. Léon Colin in his paper on 'Tuberculisation Aiguë. This view is, however, scarcely supported by the facts. I have read this paper, and find that his cases are mostly those of young soldiers who had recently joined their regiments, and M. Colin himself especially points out that they may all have been the subjects of chronic phthisis before they came out, and the change from a temperate northern climate to a Mediterranean station had produced a sudden outbreak of acute disease. He does not suggest that it was a true epidemic, and he adduces the English army statistics as bearing witness to the danger of transferring men of northern climates to hot climates.

The likeness to an epidemic is thus reduced to an accidental lighting up of the disease in an acute form by the influence of a hot climate.

Tubercle, then, can only be said to be allied to ordinary epidemic diseases in the sense that they are all parasitic disorders.

It may rather be regarded, like leprosy, as a type of an endemic disease. Dr. Guy, in his work on 'Public Health' (p. 42), when defining epidemic maladies, expressly singles out consumption as a 'non-contagious, domestic disease,' and, to point his remark, he refers to the very slight fluctuations in its rate of mortality, and says: 'If, then, anyone were to assert that this disease is contagious, which is tantamount to saying that it may be epidemic, the figures I have quoted would in themselves furnish an answer in the negative.'

It by no means follows, however, that a disease need be contagious or epidemic because it is derived from without the body, and because it afterwards travels infectiously along the channels of absorption, or of circulation. Although infective, it need not be infectious, and may be incapable of being conveyed directly from the body of one patient to that of another. The discussion of this question as regards phthisis must be postponed until later, but it may be sufficient for the present to point to intermittent fever, as an instance of a disease which, though non-contagious, is essentially infective and connected with a microbic parasite.

Whether contagious or not, phthisis is sharply marked off from ordinary exanthematous disorders, not only by its specific micro-organism and by its endemic habit, but also by its usually slow and creeping and irregular course, and by the permanent growths and alteration of struc-

tures and destruction of tissue that are brought about by the bacillus.

The exanthemata, on the other hand, are acute in their type, they run a rapid course and have certain definite and fixed periods of incubation, of inflorescence, and decline. They seldom attack the same person twice, though they are highly infectious for the most part, and after visiting a people for a time, they depart almost completely, until, in fact, a sufficient density of susceptible persons has grown up to permit them to regain their epidemic character. Lastly, they seldom leave behind them permanent growths which tend to spread destructively through the tissues.

We can thus mark off, as alien to tubercle, a large group of microbic diseases, even including those already mentioned as leaving lesions somewhat similar in character to tuberculosis, such as enteric fever and acute glanders.

In searching for the diseases kindred to tubercle we must look for those which are chronic, endemic, irregular in their course, and produced by the action of a micro-organism which is of comparatively feeble power of irritation, but which nevertheless brings about inflammatory action, tending to produce permanent changes and necrosis of the tissues, and travelling along the track of the lymphatics or in the currents of the blood.

Chronic glanders, or farcy, includes most of these points of resemblance. It is produced by a bacillus discovered by Drs. Löffler and Schütz, which has been best cultivated in blood-serum and isolated, and which produced the specific disease by inoculation into horses, guinea-pigs, rabbits, and field mice. The farcy buds and nodules caused by its presence are found not only in the nostrils, but also in the larynx, trachea, and lungs, and

they closely resemble tuberculosis in their appearance and in their structure. It has also been known to attack the nervous system. Like scrofula, it produces intractable ulcers of the skin and mucous membrane and affections of the joints. In some cases the chief symptoms are cough, with sanguineous expectoration, hoarseness, fever, and emaciation, so that it may well be mistaken for phthisis.

It is a contagious malady, but it has been found to be subject to predisposition, and susceptibility to it is enhanced by unhealthy conditions of life, impure, rebreathed air, cold and damp being especial factors in the result.

A close affinity to tuberculosis may also be traced in such diseases as actinomycosis, frambœsia or yaws, and in the fungus disease of India, or Madura-foot.

The origin of two of these disorders is, however, too obscure to allow us to catalogue them in due order. It is even doubtful whether the fungus found in the dark variety of mycetoma is essential or adventitious, and frambœsia is more acute in its course than most forms of tubercle.

Actinomycosis, however, presents many features in common with tubercular disease. It is characterised by the formation of nodules, somewhat resembling tubercular growths, and showing, perhaps, some relationship in their mode of production.

In the centre of these nodules are the specific club-shaped corpuscles, the actinomyces. As the nodule grows, a zone forms around it of large, nucleated cells, not unlike giant-cells in appearance. These are surrounded by exudation-corpuscles, and these again by a capsule of fibrous tissue. These inflammatory nodules ultimately coalesce, and gradually form large tumours, but while

still scattered and small in the lungs they much resemble tubercles. There is also in actinomycosis a greater tendency to suppuration.

Cohnheim remarks (p. 743), 'We find in actinomycosis all the essential criteria which are characteristic of the infective tumours.'

Actinomycosis also would seem to preserve a somewhat endemic character. It has been observed by Claus that in Bavaria the cattle affected by it were chiefly confined to three districts.

At present, however, it is chiefly an epizootic disease, affecting cattle rather than mankind, and, moreover, the organism causing these lesions belongs to an order distinct from the bacillus of tubercle. Technically, it is allied to the hyphomycetes, similar to aspergillus, or the pébrine of silkworms, the saprolegnia which causes salmon-disease, the oïdium of vines, and of favus, ringworm, and thrush. In spite of its infective character, therefore, and of the somewhat similar nature of its course and the injuries it inflicts, it cannot be placed in the same order as tuberculosis.

But it must be acknowledged that the closest resemblances are to be found between tubercle, syphilis, and leprosy.[1]

All these disorders are of microbic origin, they are capable of varying degrees of intensity, and possess the power of renewing their ravages after varying periods of dormancy; they produce growths that are strikingly similar in pathological characters, and they either are or have been universal in their distribution apart from considerations of climate, race, and habits of life.

Syphilis, it must be admitted, is, in certain points,

[1] Perhaps at some future time, when the microbe of cancer has been made out, this disease may be added to the group.

more like an exanthematous disease than it is like tuberculosis; thus, it is highly contagious, and is not very discriminating in its victims. It does not usually recur, it has a fairly definite period, and has a number of distinctive rashes, and in its initial stages is usually acute in its manifestations.

Nevertheless, if we can trust the researches on this point, a matter still very doubtful, it appears to be due to a bacillus, which is capable of cultivation, and which takes a special kind of stain, and this organism apparently possesses the power of remaining latent for long periods of time. It is said to have been found in all the lesions of syphilis, and these lesions, as in tubercle, are apparently due to the slow irritative action of the organism, though they are constantly more vascular in character than true tubercle. The most prominent feature of these lesions is the collection around the vessels of round cells (? leucocytes), but in the more chronic products of the disease, epithelioid and giant cells are found. The essential part of their distinctive action appears to be due to an infiltration of the tissue, and the subsequent necrosis of the part, just as we have found it to be in tuberculosis.

When the mass of inflammatory tissue is considerable, it forms tumours which, in their earlier stages, are with difficulty distinguished from tubercles. These gummata, however, differ from tubercles in being composed chiefly of translucent, gelatinous material, which, in caseating, as it does later on, becomes tough and fibrous. They have also less tendency to soften than tubercle, though they may cause destruction of surrounding tissues by ulceration, leaving a mass of scar-tissue as their ultimate result.

Their most common distribution also throughout the

body differs somewhat from that of tubercle, though when they attack the lungs the diagnosis from phthisis becomes very difficult. The virus takes its course along the same channels, which is mostly along the track of the lymphatic vessels.

But of all these disorders, unquestionably the nearest approach to tubercle is to be found in Leprosy. In their pathology, in their course and distribution, and in many other features these diseases show the closest relationship.

Perhaps I may be permitted to trace the resemblance somewhat in detail. It is probable that in certain points light may be reflected upon the character of the allied disease, and even if it should prove to be impossible to apply to tubercle the lessons taught by the disappearance of leprosy from the central parts of Europe, we shall at least be in a stronger position when we come to treat upon the preventibility of tubercle.

1. A micro-organism is closely associated with each disease, and the bacillus of leprosy is so similar in appearance, and in its reception of staining fluids, that the most accomplished bacteriologists can find little or no difference in appearance between it and the bacillus of tubercle.

It has indeed been said that the bacillus lepræ receives the stain rather more readily, but even this statement does not always hold good, and it has been proved that the human tubercle-bacillus is longer in being coloured than the artificial cultures of this same organism; and Baumgarten comes to the conclusion that, both in their morphology and their staining, they cannot be distinguished. He grants, however, that they produce decidedly different nosological results.[1]

[1] Koch points out, that 'although the bacillus of leprosy can be stained by the same method as the tubercle-bacillus, the contrary does not hold good. The former, as Neisser has shown, stains by Weigert's

The bacilli of lepra are also found in much larger proportion in the diseased tissues, than those of tubercle. Moreover, hitherto it has not been found possible to produce the disease by inoculations with the organism, although tissue-implantation has recently been successfully performed. Pure cultivations of lepra-bacillus cannot be obtained. Evidently, whilst there is a strong likeness between them, we can hardly agree with Dr. Daniellsen, who boldly says, 'On ne peut indiquer un signe certain qui permette de différencier le bacille lépreux du bacille tuberculeux.'[1]

2. Leprosy is like tubercle in its mode of attack. The entrance of the virus takes place at the most exposed portions of the body. As in phthisis the atmosphere conveys the bacillus to the air-passages, and in scrofulous glands to the nearest sore; so in leprosy contagion takes place first on exposed parts—the bare hands and face—and in countries where the feet are uncovered these are the first to receive injury. The unshod Japanese are especially liable to the disease.

Some previous injury is also necessary: the epidermal covering forms a varnish impenetrable by the parasite.[2]

3. After its entrance into the body, leprosy is usually very slow in its progress, creeping infectively through the body, often with long intervals of latency, in this respect also resembling tubercle. The main point of difference is the greater chronicity of leprosy, which lasts on the average ten or twelve years, instead of three or four, as

plan for colouring nuclei, but not the latter. So that, although the two kinds of bacilli resemble each other in form, size, &c., it is always possible to distinguish them by trying Weigert's colour-reaction.'—Syd. Soc. i. 85.

[1] *Archives Roumaines de Médecine et de Chir.* Paris: Baillieres. p. 16.

[2] Cornil and Suchard's *Microparasites in Disease.* Syd. Soc. p. 296.

in the case of phthisis. Both diseases tend towards a fatal issue, but both are capable of becoming absolutely quiescent and inert for long periods.

4. The pathology of the two diseases is also strikingly similar. Although the bacillus in leprosy chiefly affects the skin and mucous membranes, the nerves and the bones, in its tubercular forms the structure of the leprous 'knots' gives ample evidence that they are formed in the same way as tubercle, by the slow irritation of the bacillus. Baumgarten points out that the large, so-called 'lepra cells' arise through the increase of the fixed tissue-cells, whilst the lymphoid elements of the leprous growth are migrated, colourless blood-corpuscles.[1]

It is interesting to note, with reference to the site of leprosy, that it attacks internal as well as external organs, and that it has even been found in the lungs. On the other hand, tubercle-bacilli have recently been found in the nervous system.

5. Both are distinctly endemic diseases, and they are, or have been, found in almost every country where human beings are collected together in any number.

They are equally uninfluenced by climate and temperature. As phthisis is now, so leprosy was formerly, endemic, not only in Asia and Africa—where, perhaps, it originated—but also throughout Europe.[2]

[1] Pathologischen Mykologie, p. 650.

[2] Raymond, in his *Histoire de l'Elephantiase* (Lausanne, 1767, p. 106), proves that the disease existed in Lombardy in the year 603; and there is evidence that leper-houses were founded in the eighth century in France, England, Germany, and Italy. A little later Matthew Paris computed that there were 19,000 of these asylums throughout Christendom, and it is well known that in the reign of Louis VIII. of France there were 2,000 of these institutions in that country alone, which were endowed by his will. The religious orders of the Knights

I have here a map, kindly lent to me by Dr. Dreschfeld, showing the distribution of lazar-houses throughout the British Isles in the middle ages. It will be seen that they are very numerous, and so spread out through the length and breadth of the land, as to indicate a very wide prevalence of the disease.

It is probable also that many have been omitted—thus, Newcastle-upon-Tyne is not marked, and yet I have learnt from Mr. Marsden Gibson, the master of the Hospital of S. Mary Magdalene, that it was originally founded for the benefit of lepers—and, perhaps, the Hospital of St. Thomas the Martyr also. The funds of many of these places have been, for the most part, diverted to other purposes.[1]

Even at the present time its presence is sufficiently manifest in the four quarters of the globe to show that it is independent of climate.

It will be seen from the map of the world that has been drawn by my friend Dr. Dreschfeld, who has kindly lent it to me, that, in Europe, it exists in somewhat large proportions in Norway, and on the Russian coasts of the Baltic and the Gulf of Finland, and on the shores of the Black Sea. It is abundant in Roumania and parts of Turkey and Greece, in the north of Italy and on the Riviera; in Sicily and on the coasts of Spain and Portugal. Iceland is also deeply stained with the disease taint. In Africa, it will be seen to exist around almost the whole of its vast coast. It is widely spread in Morocco, Tunis, Tripoli, Egypt, and Abyssinia;

Hospitallers, and especially those of Saint Lazarus, were founded mainly for the care of lepers, and their chief was usually himself affected with the disease.

[1] Since this lecture was delivered Dr. Dreschfeld has received several communications from medical men indicating other sites of leper hospitals, and these have now been added to the map.

BURY
WINSLOW
BALDOCK
DUNSTABLE
COLCHESTER
AYLESBURY
OXFORD
ST ALBANS
MALDON
BERKHAMSTED
HIGH WYCOMBE
HIGHGATE
LONDON
SADING
DARTFORD
ROCHESTER
CHATHAM
CANTERBURY
DOVER
ROMNEY
HYTHE
CHICHESTER
HASTINGS
BRIGHTON

THE LEPRA HOSPITALS.

OF THE

BRITISH ISLES.

DURING THE MIDDLE AGES.

Prepared by Dr J. Droschfeld
Professor of Pathology
in the Owens College Manchester

GEOGRAPHICAL DISTR
AFTER L
ARCTIC OCEAN
GREENLAND
NORTH AMERICA
Lawrence
ATLANTIC
MADEIRA
OCEAN
PACIFIC
OCEAN
SOUTH AMERICA
PREPARED BY DR J. DRESCHFELD
PROFESSOR OF PATHOLOGY
IN THE OWENS COLLEGE MANCHESTER

BUTION OF LEPROSY.
LOIR.

at the Cape and in Madagascar, and Upper and Lower Guinea.

In America, it exists in Greenland, Quebec, and in many of the northern and most southerly states, Alabama, New Orleans, and Texas. It is widely spread in Mexico and California. In South America the northern and eastern portions are widely stained as far south as Monte Video.

In Asia, Arabia, Persia, Turkistan, and the coasts of China, Siam, and Japan are all more or less tinctured with it; but India is its great hotbed. In Australia, Queensland on the north, and New South Wales and Victoria in the south, are affected by it, and both Tasmania and New Zealand are darkly tinted.

It seems to have a special predilection for islands; thus, in addition to those I have already mentioned, the Grecian Archipelago is especially subject to it: Crete, Tenedos, Patmos, and Rhodes; Samos worst of all. It is found also in Cyprus. In Asia, the Philippines, the Moluccas, the Celebes, the Caroline Islands, the Mariannes, Andamans, and Nicobars, the isles of the South—Borneo, Sumatra, and Java; off America the Antilles, the West Indian islands; off Africa, Madeira, the Canaries, the Cape de Verde islands, Socotra, the Isle of France, the Seychelles, St. Helena; and Robbin Island, off the Cape of Good Hope, is made into a leper sanatorium.

In the Pacific, we have only too recently heard of its ravages in Hawaii, and it exists, though less abundantly, in Tahiti, Fiji, and New Caledonia, where it seems to have been recently introduced.

It is obvious, from this long list of habitats of the disease in the past and present, that, like phthisis, it is independent of climate, and, though certain races are more prone to it than others, none are entirely exempt.

It is also more common amongst the poor and filthy than amongst the rich and cleanly; but it is not confined to this class, and appears amongst the well-fed as well as amongst those who are confined to vegetable diet, and those who feed upon fish.

6. Precisely the same controversy as is now waged with regard to phthisis, is carried on as to the contagiousness of leprosy. In early times and the middle ages, in all countries, no doubt at all was entertained upon the point. In all Eastern countries, the leper was 'unclean,' his clothing and dwelling were equally regarded as contaminated, and in Europe all kinds of devices were adopted to avoid contagion.[1]

This belief in the contagious nature of leprosy, still remains in many countries, and lepers are carefully isolated in Norway, though without excessive rigour.

But the greatest differences of opinion prevail on this question amongst medical men.

In the report of your own College in 1860, the Commission state decidedly, 'that the weight and value of the evidence they furnish is very greatly in favour of the non-contagiousness of leprosy' (p. vii), and again, 'the all but unanimous conviction of the most experienced

[1] He wore a distinguishing costume, and carried a bell or clapper with which to warn those he met. He had a separate 'borde' or hut, or slept under a hedge; he might not even look into any well or fountain, or drink from any stream but his own; must keep to the leeward of anyone whom he might meet or speak to. He had to wear gloves when he passed over a bridge, and could go nowhere without a special licence ('Curiosities of Leperdom,' *Cornhill Magazine*, August, 1889, p. 143, quoted from Grosley's *History of Troyes*). In the fourteenth century, they were supposed to be associated with the Jews, in a horrible plot to poison all the springs, wells, and rivers with their blood; and in 1321 a fearful massacre of them took place in France. At Chinon 160 were burnt in one day, and in Périgord and Languedoc, in a plague panic, fires were lighted everywhere, and lepers and Jews heaped thereon.

observers, in different parts of the world, is quite opposed to the belief that leprosy is contagious or communicable by proximity, or contact with the diseased.' Just as we shall see presently, in the case of phthisis, they quote the experience of hospitals, and the immunity of the attendants in leper asylums.

In the discussions on this question at the International Congress at Copenhagen in 1884, the two chief exponents of the disease, Dr. Zambuco of Constantinople, and Dr. Hansen of Christiania, took entirely opposite views; the former, maintaining that leprosy is not contagious, but is hereditary, and the latter, that it is contagious and not hereditary. Dr. Daniellsen, the father of research on the subject, also takes the non-contagious view, and says, 'According to my observation, heredity is sufficiently proved, whilst I have not found one single well-proved case of infection.' [1]

7. A similar difference of opinion is to be found as to the influence of heredity in both diseases. With regard to leprosy, Dr. Hansen states that of 160 Norwegian lepers who had settled in the states of Wisconsin, Minnesota, and Dacota, none of the offspring, in some cases as far as the great-great-grandchildren, have shown signs of the disease. He points out that there could have been no question of heredity in the case of the great outbreak of the disease in Hawaii.

A recent writer on the subject, Dr. Albert Neisser, also considers that leprosy is not a hereditary disease, though he acknowledges that the susceptibility of the individual appears to be of more importance than in any other bacterial disease.

It is not surprising that, with all these points of

[1] See paper in *Monatshefte für praktische Dermatologie*, 1885, by Dr. A. Wolff, of Strassburg.

similarity, the question should be asked, as it is by Daniellsen, whether 'leprosy itself is not a tuberculous disease.'

'The circumstance that the bacilli of tubercle are larger and less disseminated in the tissues,' he says, 'ought not to be invoked as a sufficient diagnostic characteristic. Nor can we draw any more conclusive argument from the mode of development of these bacteria.'

'The manner in which infection in leprosy takes place, the long and silent period of incubation which characterises it, recalls in the highest degree the infection of tubercle.' [1]

Dr. Daniellsen further affirms that tubercular disease is more common amongst lepers than amongst the general population; but, even if this were true, the fact might fairly be ascribed to the general low state of health of lepers making them more vulnerable to the attacks of the tubercle-bacillus.

This statement is, however, contradicted by Dr. A. Hansen, who, as I am informed by Dr. Stabell of Bergen (in answer to a question of mine), states that 'Tuberculosis, which was formerly very prevalent in the leper hospitals of Norway, (and no doubt the patients were infected in the hospitals), is now more seldom seen, because the hospitals are now not so crowded as before. Tuberculosis indeed attacks lepers as it does other people, but the acute form is not more frequently seen amongst phthisical lepers than amongst other persons affected with phthisis.'

On many grounds, therefore, the doctrine of identity must be dismissed. The different behaviour of the two kinds of bacillus in the presence of cultivating media, their selection of certain tissues as their chief haunts,

[1] *Loc. cit.* at p. 23.

the difference in their rate of progress, and the fact that there is no special tendency towards tubercular disease amongst lepers, and *vice versâ*—all these considerations show that, though they are certainly closely related, yet there is no identity to be discovered between them.

At this stage it may be useful to draw certain practical conclusions from this review of the pathology and affinities of tubercle; they may assist us when we come to deal with the question of its preventibility:—

1. That the bacillus which provokes the disease comes most frequently from outside the body, and is conveyed by means of the atmosphere.

2. That it requires a certain length of time, and certain narrow limits of temperature within which it can develop.

3. That when it is entrenched within its encapsulating tubercle it is placed at a distance both from the currents of blood in the vessels, and from air entering through the lungs.

4. That its life in the body is usually a short one, and that the living tissues are antagonistic to it, or, to use Dr. Moxon's words, that 'the life of the bacillar parasite is difficult, easily discouraged by unfavourable circumstances, like an aphis by an easterly wind;' and

5. That the resemblance which it bears to the bacillus of leprosy may encourage us to hope that, as this latter disease has been banished from our country, so also may the different forms of tuberculosis ultimately disappear.

LECTURE II

THE TOPOGRAPHY AND DISTRIBUTION OF PHTHISIS

Ubiquity of phthisis—Influence of climate—Variations in countries; in counties—Male and female phthisis-rates—Areas of immunity: (*a*) in sparsely-populated regions; (*b*) elevated sites; (*c*) sub-arctic regions—Influence of subsoil—Malnutrition—Hardships—Exposure to the elements—Occupation—Dusts—Stooping and constrained postures—Variations of temperature—Ill-ventilated workshops—Re-breathed air—Tubercular infective areas—Infected houses.

It is not unreasonable to expect that a study of the geographical distribution of phthisis would lead us to the discovery of at least some of the fostering causes of the disease.

In the different countries of the world, the racial differences of the inhabitants are often so great, and the conditions under which they live, as to climate, food, habits, &c., are so varied that we might naturally expect to find some nations suffering more than others, and hence we might be able to lay our finger upon the peculiar circumstances that have been either favourable or adverse to its progress.

Nor are these anticipations entirely discredited by the result, though a much more thorough examination of statistics is required than might at first appear to be necessary.

Our chief sources of information, as to the comparative prevalence of phthisis, are the two great general works, Hirsch's 'Geographical and Historical Pathology,' and

DISTRIBUTION

RATE PER THOUS

N OF PHTHISIS.

ISAND OF LIVING.

Lombard's 'Climatologie Médicale;' but we are able to obtain some data from the 'British Army and Navy Medical Reports,' from official statistics of disease in different countries, and from various works by individual medical men.

Lombard's comparative figures are not very satisfactory, as he adopts the fallacious method of comparing the number of deaths from phthisis with each 1,000 of the total deaths, a method that gives a lower ratio from consumption the higher the general death-rate; and thus a place with double the rate of mortality from phthisis, as compared with another with the same population, may appear in the same grade with it, if only its total death-rate is also twice as great as that of the latter.

Many of the other statistics are also open to objection; but in a general survey, such as we propose to make, it may, perhaps, be permissible to make use of somewhat imperfect materials.

Hirsch's tables are the most trustworthy, as he gives the mortality from phthisis per 1,000 living, and the years to which the returns apply, together with his sources of information, which are always the best that can be obtained. I have thrown his principal figures into the form of a diagram, which exhibits graphically the variations in the complaint better even than the figures themselves.

I have also a table drawn from Dr. Lombard's work, which shows the proportionate mortality in the capitals of the world.

TABLE II.—*Proportion of Deaths from Consumption to* 1,000 *Deaths at*

London	121	Rome	114
Paris	143	Milan	132
Brussels	163	Lisbon	115
Vienna	208	Athens	183
Berlin	109	New York	167
Stockholm	160	Rio de Janeiro	186
Christiania	172	Lima	171
St. Petersburg	151		

At the first glance at the figures placed at our disposal from these various sources, it would seem as if the expectations with which we commenced our quest were doomed to disappointment.

The disease appears upon the death-roll of almost every nation; it is everywhere present in greater or less abundance; and even if some countries appear to suffer less than others, certain places within their boundaries are to be found in which the disease exists in more than average proportion.

As both Hirsch and Lombard remark, 'Phthisis is a ubiquitous malady.' If we look at the map of the world which the latter provides, in which various degrees of shading are made to denote the presence of the malady in greater or less abundance, it would appear that he is quite justified in his assertion. In all climates, in the north, and in the south, east, and west, however various the conditions under which men live, however much they may differ in race, in diet and clothing, and in habits of life, wherever human beings are congregated together, there phthisis is to be found.

With very few exceptions, the degrees of shading denoting variations of prevalence serve also to denote variations in the relative density of the population in different parts of the globe. In other words, in proportion to the number of the people aggregated together upon a given space of ground, so also is the rate of mortality from phthisis.

In Dr. Lombard's maps, the only portions of the earth's surface from which the colour is entirely absent, are the arctic and sub-arctic regions, deserts, and high ranges of mountains, and it is precisely in those parts that human beings are fewest and most sparsely scattered over the ground.

In all the capitals of countries, and in the chief cities of Europe, Asia, Africa, and America, there is but little difference in the phthisis rate, and what differences there are are not to be accounted for by differences in climate. See Table II.

We are impelled to similar conclusions by a survey of the army and navy returns from all parts of the world.

We might then hastily conclude that but little of any value can be obtained from such a research as this, except that the very universality of the disease connects it with the collection together of human beings. And such a conclusion might, perhaps, have been reached by *à priori* reasoning. In the light of our present knowledge of the essential cause of phthisis, there is nothing surprising in our thus finding the disease wherever human beings are gathered together. So far as we know, the chief source of the organism that provokes the malady is the human race, and hence, unless there are adverse conditions, it might be expected to follow mankind in its distribution over the surface of the globe. Without further investigation then, it would appear as if the organism could exist under all conditions under which human beings could also live, and as if our search for secondary causes would lead to no definite or certain result. But this view would certainly be an erroneous one. It is already something to have ascertained that the disease *may* exist everywhere, under every sky, in country or in town, in all classes of society, in the palace and in the hovel, amongst the luxurious and well-fed, as well as amongst the poor and starving, but much more than this can be deduced from the statistics at our disposal. If we break up the figures presented to us into smaller groups—in other words, if we try to analyse the returns of the disease—we shall find out that it has

its predilections; and by observing more narrowly its selection of favourite habitats and breeding-grounds, we shall obtain important indications that will guide us to a discovery of the conditions that determine its choice.

1. We have already some reason, in the almost universal presence of the malady, to conclude that the ancient doctrine of its origin in a damp, changeable climate is erroneous. It has been called 'la maladie Anglaise,' from its supposed excessive prevalence in this country; but we have already seen that it prevails to a still greater extent in the dry, sunny atmosphere of Egypt, as at Alexandria; and in the hot climate of South America, as at Pernambuco and Rio de Janeiro; but we may take another method of proving its independence of climate.

Let us take the extreme variations in the disease, in places geographically close together. We shall find that they are so great, and so frequent in different countries, that they could not have been due merely to differences of climate.

If we take the map of almost any country in the world and shade its several provinces so as to denote the varying prevalence of the disease, we shall discover not only that a great variety of tint has to be used, but also that the dark parts alternate with the light, without any reference to geographical position, north or south, east or west.

Thus, in Dr. Haviland's map of the distribution of phthisis in England and Wales, we find that dark blue and dark red spots, the antitheses of prevalence, are interspersed all over the map.

The same observation is true of Boudin's maps of France, showing the prevalence of chest-disease and scrofula. I have here also a map of Norway, display-

ing the same alternations of light and dark in all parts of the country.

But we may come to closer quarters still, and may take the variations within the areas of counties instead of countries ; for instance, within the counties of England.

The following table shows some of these variations in different parts of England, and care has been taken to avoid the mention of places whose returns might be vitiated from the presence in them of large hospitals. There are only two exceptions to this rule, namely, Hastings in Sussex, and Liverpool in Lancashire. It will be noted that, in most of the counties represented, the difference between the highest and lowest returns is more than 50 per cent, and in some the highest reading is more than three times that of the lowest.

TABLE III.—*Variations in the Consumption-rate, at ages* 15–55, *per* 100,000 *living at those ages* (*Males*)

Counties	Towns	Highest	Lowest
Surrey	Guildford	526	—
	Farnham	—	242
Sussex	Hastings	(H)643	—
	Battle	—	180
Oxford	Headington	566 [1]	—
	Banbury	—	241
Cambridge	Whittlesea	469	—
	Wisbeach	—	230
Norfolk	Walsingham	439	—
	Flegg	—	257
Wilts	Salisbury	438	—
	Mere	—	201
Cornwall	Redruth	461	—
	Launceston	—	223
Lincoln	Spilsby	361	—
	Caistor	—	205
Lancashire	Liverpool	(H)602	—
	Wigan	—	249
York	Reeth	589	—
	Settle	—	253

[1] Since this lecture was delivered I have been informed that the Radcliffe Infirmary, Oxford, is in Headington Union.

The same variation is to be found in other parts of the United Kingdom. Thus, in Scotland, consumption is almost unknown in the Western Hebrides, but, in towns on the west of the mainland, with a very similar climate and a similar race of people, it is very common. In Edinburgh, the rate per 1,000 deaths[1] is 102; in Glasgow, 371.

In Ireland, again, the rate of mortality from the disease per 100,000 of the population in the ten years, 1865–1874, was in the eastern division 259·62; in the western, 95·64.

Similar variations are to be observed in other parts of the world. Lombard gives the number of persons dying of consumption per 1,000 deaths, in different places, as follows:

In Canada, the numbers vary from 241 in New Scotland, to 138 in Quebec. In Russia from 190 in Archangel, to 30 in Riga. In Holland, from 141 in La Dreuthe, to 64 in Zealand. In Belgium, from 240 in Limbourg, to 122 in Luxembourg. In Germany: Dresden 147, Weimar 74. In Italy: Milan 132, Turin 83. In Portugal: Lisbon 115, Malaga 54. In Egypt the general rate is 25, in Cairo 101. The variations cannot be due to climate, and probably arise from differences in the density of the population and in their mode of living.

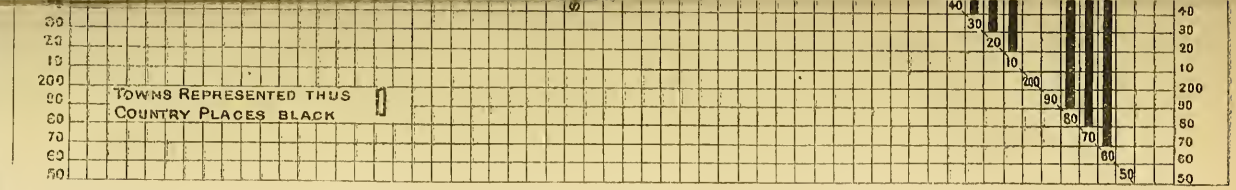
But there is another mode of analysis possible. Perhaps the strongest proof of the existence of causes much more powerful than climate in producing a tendency to consumption, is to be found in the great differences between the male and female rates of mortality from the disease, in the same towns and districts. In the following table, I have selected from 'Lowe's

[1] Lombard.

Male and Female Phthisis Rate.

In Town and Country Places.

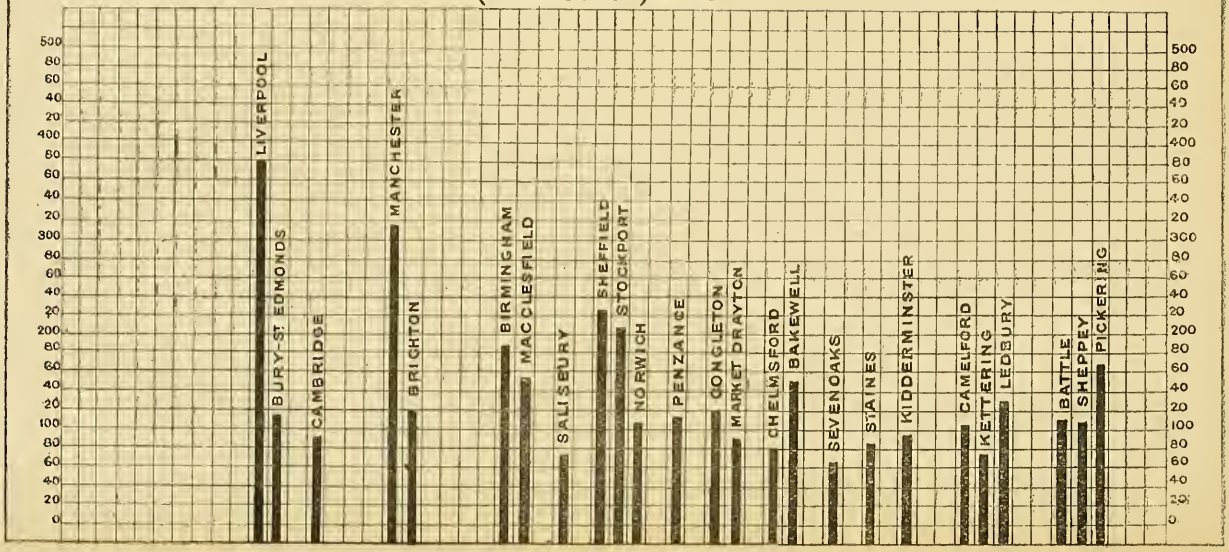
In this diagram the extent of the male phthisis-rate is shown by the position of the place o
above the diagonal line when the female



DISEASES OF RESPIRATORY ORGANS, (NOT PHTHISIS.)

DEATH RATE PER 100.000 LIVING

(AT AGES 15-55) MALES



diagonal line; the differences between the male and female rates by the length of the vertical lines—
is the greatest, below it when it is least.

Tables ' the male and female rates—(1) in places where the male rate exceeds the female rate, and (2) in places where the female rate is much the highest.

TABLE IV.—*Annual Average Proportion of Deaths from Consumption between the ages* 15 *and* 55 *per* 100,000 *persons living at those ages between the years* 1861–1870.

Towns	Males	Females	Differences
(1.) Cambridge	570	395	− 175
Whitechapel	560	430	− 130
Bath	540	255	− 185
Greenwich	525	375	− 150
Brighton	520	345	− 175
Southampton	500	385	− 115
Birmingham	475	345	− 130
Newcastle	470	395	− 75
Salisbury	440	305	− 135
(2.) Sedbergh	365	615	+ 250
Congleton	360	595	+ 225
Bootle	225	555	+ 330
Leek	355	525	+ 170
Belper	275	455	+ 180
Buckingham	275	455	+ 180
Sevenoaks	290	455	+ 165
Alston	285	425	+ 140
Camelford	230	295	+ 155
Battle	180	365	+ 185
Pickering	160	315	+ 155
Billesdon	120	265	+ 145

The differences between these figures, concerning two groups, male and female, of the population of the same places, constitute the main points of my argument at present, but I shall presently have occasion to return to them and attempt to draw from them the other lessons that they teach. The accompanying diagram shows graphically the differences between the male and female rates in towns and country places.

Enough has now been shown, from the details of the statistics at our disposal, to prove that we cannot

find in mere climate enough to account for the varying prevalence of consumption.

We have yet to consider the causes of the existence, upon the earth's surface, of cases of immunity from the disease. We have seen already that there are large tracts of country that are nearly, if not quite, free from the disease, and although this immunity may be due to sparseness of population, it is reasonable to ask whether an analysis of the figures may not bring to light some climatic or other conditions less favourable to the disease.

In Lombard's maps we have seen that there are certain broad spaces, untinted with colour, showing regions still untainted by the disease. These spaces may be grouped broadly, under three heads:—

1. Deserts, or sparsely populated places.
2. Mountain ranges or high plateaux.
3. Arctic, or subarctic regions.

It is not surprising to find that consumption is rare in the first of these groups. Where there are few human beings it is hardly likely that there will be much disease. But it is not merely the scarcity of possible victims that makes the disease uncommon, for the proportion of cases to the number of inhabitants in these districts is also very low, so far as we can judge from the imperfect data at our command. Thus we are told by both Lombard and Hirsch that there is almost complete immunity from the disease in Nubia and Upper Egypt, that it is very rare in Abysinnia and Central Africa; in the capital town of Oran, with 25,000 inhabitants, it is practically non-existent; and Livingston states that it is quite unknown in South Africa and in the interior of Cape Colony. It is rare in Asia Minor, Mesopotamia, Syria, Arabia, and Persia. In the steppes of Tartary, the interior of Australia, and in Tasmania it is almost unknown.

There are, however, certain considerations, apart from the wide separation of these districts from one another, that prevent us from ascribing this freedom from disease altogether to the climate. These spaces are often bordered by regions where the disease is much more frequent. Thus, in Asia Minor, it is often met with on the coast or in the principal towns. The Bedouins on the coast of the Red Sea, 'who exchange their tents for stone-built houses,' suffer from consumption.[1] In Syria, it is met with at Aleppo, and in the Soudan at Khartoum. In Zanzibar it is said to be especially common among Arabian women of the higher class—perhaps owing to their greater seclusion. Whilst it is rare amongst the native Persians, who live an almost open-air life, it is more common amongst foreigners. In Algeria, whilst the nomad Arabs are free, 'amongst the captives many die from the disease;' and in Egypt it is noted that whilst Syrians, Turk, Armenians, and Europeans seldom contract the complaint, Jews often become scrofulous, and die frequently of consumption. At Alexandria the mortality is nearly double that in England. In Australia it is almost as common in the large towns as it is in Europe. Again, we have to note the fact that in many parts of the world formerly almost free from the disease it is now rapidly increasing in prevalence. Information to this effect comes to us not only from Australia, but from Greece, Brazil, New Zealand, and the United States of America. Some observers state[2] that consumption is unknown in the western prairies of Illinois, Missouri, Iowa, Wisconsin, and Utah, and also in the Rocky Mountains; but, according to others, it has of late begun to show itself much more frequently, especially amongst the female immigrants.

[1] Hirsch, vol. ii. p. 65. [2] Hirsch.

The climate has not changed, but the conditions under which life is carried on, especially the crowding together of the inhabitants, have altered, hence the increase of the disease. As Hirsch remarks,[1] 'an altered mode of life amongst whole populations has had a most decided influence upon the rise and progress of consumption, whilst the climate in which they live remains the same as it was before.

INFLUENCE OF ELEVATED SITES

The second group of districts free from consumption, or nearly so, is of those parts of the earth elevated to a certain height above the level of the sea. In Lombard's map of the distribution of the disease, all the great mountain ranges are left blank:—the Dovrefeld, the Ural mountains, the Alps and Apennines, the sierras of Spain, the Carpathians, the Hartz, the Grampians in Scotland; in Asia, the high plateaux of Armenia and Persia, the Himalayas, and the mountain ranges of China; the table-lands of Abyssinia and of Mexico in North America, and the table-lands of Quito and of the Andes in the south continent. In all parts of the world, then, and in all climates, an elevated site seems to be, to some extent, a safeguard against consumption. The degree of elevation conferring this immunity was supposed by Gastaldi (who was one of the first to notice it) to be over 600 mètres—about 2,000 feet—the disease being as common at points just under that height as on the plains. But in Western Texas, at a height of 2,100 feet, consumption is not uncommon; and the same observation has been made respecting the elevated plains of Castile and Leon, and of the Styrian highlands. It has been conjectured also that the freedom from the disease was

[1] Vol. ii. p. 89.

due to the greater rarity of the atmosphere at these heights;[1] but it must be remembered that the population of these elevated regions is much less dense than that of the lower ground, and that the air in consequence is much less polluted with organic matter. Mountain and sea air are notoriously much freer from organisms than the air of plains, and Dr. Henry Bennet ascribes the beneficial influence of the climate of Mentone to the fact that the air breathed by the inhabitants must come either off the sea, or else over a range of mountains 4,000 feet in height. That the immunity is not simply due, however, to the rarity of the air is completely proved by Dr. Emil Müller, who, in his treatise on the 'distribution of consumption in Switzerland,' shows that there is no complete immunity from the disease in that Alpine country. A certain proportion of the inhabitants of these high places die of the disease, the rate depending, not upon the elevation of their dwellings above the sea, but upon the nature of their occupations. Industrial *indoor* pursuits give a rate varying from 6·5 to 10·2 per cent., and one of the highest of these rates (9·8) is at an elevation of 3,400 to 4,400 ft. At 4,400 to 5,000 ft., in mixed labour, the rate from the disease was 7·7 per cent. Here, then, we are obliged again to fall back upon the conditions of social life as a much more powerful factor than the influence of climate or locality.

[1] Dr. Marcet has recently carried on a series of observations upon the influence of altitude upon human respiration, which may throw some light upon the advantage of high levels in the treatment of phthisis ('Proc. P. S.' vols. xxvii. xxviii. xxix. xxxi. and xlvi.) He proves that the volumes of air required to yield the oxygen necessary for the production of a given weight of carbonic acid, are smaller on mountains under diminished pressures, than in the plains under high pressures (the mean difference was 12 per cent.); and he concludes that the respiratory function is therefore carried on more perfectly at these heights than in the plains.

INFLUENCE OF SOIL

Closely allied to this point is the question of the influence of soil upon the disease. It has been shown by Dr. Bowditch in America, and by Dr. Buchanan in England, that places situated upon a damp impenetrable subsoil are much more subject to consumption than those placed upon a porous soil. Good drainage also has been found to diminish the prevalence of the disorder by as much as 50 per cent. These results have since been confirmed by Dr. Haviland, and by the Registrar-General of Scotland.

In the conclusions drawn from his map of the distribution of phthisis in England and Wales, Dr. Haviland says: 'Damp, clayey soil, whether belonging to the wealden, oolitic, or cretaceous formation, is coincident with a high mortality;' and the Registrar-General, in his seventh report, remarks that 'the towns, villages, hamlets, or houses which were situated at or near undrained localities, or were on heavy impermeable soils or on low-lying ground, and whose sites were consequently kept damp, had a very much larger number and proportion of cases of consumption than towns, villages, hamlets, or houses which were situated on dry or rocky ground, or on porous light soils, where the redundant moisture easily escaped.'

In an address to the Sanitary Congress, held at Leicester, in 1885, I gave the details of an inquiry I had myself made into this subject, that goes even further than those already cited—a contrast between two populations, one being on clay lands, the other on a hill of sand. The result was derived from a ten years' mortality table, and was that, whereas in this period there had originated twenty-two cases per 1,000 inhabitants on the

clay lands, on the sand only one per 1,000 had thus suffered, and that not one of the children or females of the population who were constantly resident there had contracted the disease. In this instance, however, we had not only the influence of a dry soil to deal with; the houses were those of well-to-do people, and were fairly well-ventilated. Whether there would have been the same immunity under other conditions is very doubtful. Still it is evident that there is a close relationship between the condition of the soil and consumption, a relationship so close that, as we have seen, a residence on a porous soil, under otherwise favourable hygienic conditions, will apparently preserve the whole community from the disease.

It is further noticeable that in these cases hereditary predisposition makes no difference in the result. There were present in these populations many whose parents or near relations had died of the disease, and yet they did not contract it so long as they lived in the place.

And yet there is no necessary connection between dampness of soil and consumption. Dr. Buchanan's experience with respect to the influence of drainage of towns has not always been confirmed by other observers.[1] Thus, 'at Ashby-de-la-Zouch, the mortality from phthisis rose 19 per cent. after the ground was drained.' In Brunswick, according to Reck, 'the mortality from consumption has not been greater in the wet parts of the town than in the quarters on a dry soil.' 'In Dantzic, where a system of main drainage was carried out fully in 1871, the death-rate from phthisis, which had been (according to Lievin) 2·12 per 1,000 in the eight years

[1] Dr. Kelly, M.O.H. for East Sussex, has shown that in 1861–70, the mortality of the several districts named by Dr. Buchanan had changed from that of 1851–60 without any difference of drainage.

preceding (1861–70), rose in the nine years following (1871–79) to 2·48 per 1,000.' 'At Berlin, in like manner, no notable effect on the prevalence of phthisis can be traced to drainage of the ground,'[1] and Hirsch says, 'It seems to me to be a more probable explanation that other ætiological factors, besides the influence of soil, come into the account under the given circumstances—factors that have a modifying effect upon the amount of the sickness, and serve to neutralise the benefits even of the most favourable conditions of soil.[2]

Moreover there are, in different parts of the world, large tracts of country that are excessively damp, many of them indeed wholly given over to malaria and to intermittent fevers, and yet these districts are so free from the prevalence of consumption that some Continental writers have supported the view that ague is antagonistic to phthisis. This theory has been conclusively proved to be erroneous,[3] but the different behaviour of the disease in different districts would of itself be sufficient to show that the connection between dampness of soil and tubercular disease is not essential, and that the influence is one simply of predisposition, and favourable only to the development of the micro-organism when introduced by other means.

INFLUENCE OF COLD

The last group of districts, to a great extent exempt from consumption, consists of the arctic and subarctic countries of Greenland, Labrador, Iceland, Spitzbergen, Nova Zembla, Finland, Siberia, Canada, and the northern parts of the North American continent.

These are all cold countries, and it is owing to the geographical position of this group of districts, that we

[1] Hirsch, pp. 203–4. [2] *Ibid.*, p. 204. [3] *Ibid.*, ii. 99.

are unable to say that climate is wholly without influence upon the disease.

We do not know why extreme cold should be thus antagonistic to consumption, and the fact is entirely opposed to the notions of our forefathers on the subject. Not many years ago it was supposed that cold and exposure to the weather were amongst the most potent of the causes of consumption. Patients were sedulously guarded from every breath of cold air in well-warmed rooms, or they were sent to warm, relaxing climates, such as those of Montpellier or Madeira.

We understand now somewhat better the operation of bright, dry, sunny atmospheres, and we know that their beneficial influence arises rather from their tempting the poor consumptive invalid into the open air, than from their freedom from cold.

It is now certain that not only is there comparatively little consumption in cold climates, but that many people predisposed to the disease, or actually consumptive, derive great benefit from a sojourn in the keen, dry, cold air of Canada, or from a winter spent amidst snow and ice at Davos Platz or the Engadine.

But it is not yet clear in what way a cold climate exerts this favourable influence; we may indeed surmise that the lower amount of humidity in the atmosphere of these regions may have something to do with the results. Cold air has a much smaller capacity for aqueous vapour than warm air. It may thus be less capable of sustaining the life of the microscopic organisms that are the exciting cause of consumption; or again, it is possible that there are smaller quantities of organically charged vapours arising from the ground, frozen as it is for so large a portion of the year. Moreover, in a frosty air the condensed moisture may entangle the organisms in

its meshes, and may carry them down out of harm's way.

But all these suggestions are mere guesswork, and there are as yet no certain proofs as to the mode in which the tendency to consumption is lessened in these regions. Here again, also, we must notice the circumstance that all these countries are very sparsely populated, and that there is, therefore, comparatively little respiratory impurity constantly floating in the air. Moreover, just as it was found possible for men to override the beneficial influences of an elevated site, so, in cold climates also, they may live under conditions such as will lead to consumption as certainly as in less favoured countries. Thus we are told by arctic voyagers, that the Esquimaux are not seldom affected by consumption, and the fact is hardly strange when we consider the mode in which they exclude all the air they can from their crowded huts. In Greenland, phthisis is one of the commonest causes of death, and similar reports come from New Archangel and Alaska. In Canada also at one time the soldiers sent to that station died of consumption at three times the rate of the ordinary civil population in England in healthy districts.

The mortality from this disease amongst the troops in Canada, in the years 1830 to 1837 was no less than 23 per 1,000 of strength; and in healthy districts in England and Wales, at ages corresponding to those of the soldiers, the rate was only 7·7. In all England the rate was 9·2, and in Manchester, with all its unfavourable surroundings, it was 12·4, little more than half the military rate in Canada. Tne cause of this heavy mortality was discovered to be bad drainage, and want of proper ventilation of the barracks; and after these defects had been remedied, from 1863 to 1872 the rate was

9·49, and in 1874 it was only 6·0. These facts again point to other conditions of much more importance than that of climate alone.

2. We may next inquire statistically into the influence of *Malnutrition*. It is not surprising that, at one time, tubercular disease should have been ascribed to some form or other of bad nutrition, and especially to a deficiency of fatty food. The poor ill-nourished denizens of towns have always furnished the largest proportion of victims of the malady. Thus it was found by M. Marc d'Espine that at Geneva, in every 1,000 deaths amongst the poor, 233 were from consumption, whilst of the rich, only 68 were from this disease.

And there are still medical men of eminence who ascribe to this cause the chief part in preparing the body for the reception of the bacillus.

Dr. Jaccoud, in his work on phthisis, affirms that the consumptive constitution is essentially due to 'insufficient nutrition, taking this word in its widest sense.'

M. Bouchardat also, in his treatise on hygiene, published in 1883, affirms emphatically that 'the continuous loss of calorific elements, in any considerable proportion, leads to pulmonary tuberculosis' (p. 665). He defends this opinion by reference to the large mortality amongst cows, from tuberculosis, when the supply of milk is forced, and by the appearance of tuberculosis in the lungs of diabetic patients, when the elimination of sugar has taken place to any considerable extent for a sufficiently long period (p. 653).

He even ascribes the 68 deaths, in Marc d'Espine's tables among the well-to-do, to some form of '*Misère physiologique*.'

No one can doubt that want of sufficient food and mental distress are powerful agents in preparing the

ground for the successful sowing of the seeds of consumption, but even if we had not known of the existence of a specific organism, and indeed before it was known, statistics were forthcoming to show that these causes are by no means essential to the production of the disease.

The terrible mortality from phthisis that prevailed at one time amongst the finest soldiers of the British army was certainly not brought on by starvation or misery. It occurred for the most part when they were not on active service, but in a time of peace, when they were well fed and well cared for so far as their bodily comfort was concerned—far better, in fact, than the half-starved workpeople and labourers, who only died of the disease at one-third the rate that they did.

3. Similar remarks, so far as the army and navy are concerned, would apply to the next series of supposed causes of consumption, namely, *hardship, exposure to the weather*, and cold. Nay! these conditions might rather be said to be antagonistic, than favourable to the onset of the disease.

The poor fishermen of Iceland, the hunters and trappers of North America, the nomad tribes of Asia and Africa, the wretched natives of Australia—all these people escape the disease almost entirely, whilst one-third of the deaths of the well-protected, well-clothed adult inhabitants of towns are from this cause.

The Highlanders, who inhabit well-built houses on the mainland of Scotland, are subject to the same fate as the other inhabitants; whilst the ill-fed, ill-clothed fishermen of St. Kilda and the Hebrides, who are of the same race, hardly ever contract the disease.

A striking case in point is quoted by Dr. H. Bennet from Professor Hind. 'Consumption,' he says, 'is unknown amongst the natives of Labrador, whilst they remain in

their own country. Here they live a kind of wild life, in tents made of spruce branches, imperfectly lined with skins, and more or less open on all sides to the air. 'They are exposed to famine and all kinds of hardship; but when they come down the great river, St. Lawrence, to take part in the fisheries, they occupy well-built houses, and being well-paid, they live in comparative luxury, and then in the course of a year or two become consumptive, and thus miserably perish.'

We shall presently have to inquire into the question, how far inflammations of the respiratory apparatus constitute a predisposing cause of consumption; but, whatever may be the outcome of that inquiry, it scarcely affects the present issue. It is very doubtful whether exposure to the elements, taken alone, is a common cause of these inflammatory attacks. We do not find that men who are much in the open air, and exposed to all vicissitudes of weather, are especially prone to catarrh or affections of the lungs. Soldiers on campaign, sailors, fishermen, hunters, gipsies, engine-drivers, coachmen, gardeners, agricultural labourers—none of these people suffer much from colds, unless they are intemperate. Arctic explorers confront the chilliest air that ever flowed. We are told also that Whymper slept safely '*sub divo*, in chill Alaska, with only a screen to windward, when the mercury in his barometer was frozen hard, and Von Wrangel relates quite a similar experience in respect of the dwellers by the shores of the Arctic Ocean.'[1]

The experience of volunteers, who are generally men unaccustomed to open-air life, is to the same effect. On the other hand we know that the inhabitants of towns not only contract diseases of the lungs, but die of their consequences in excessive numbers. It has been

[1] McCormac, *Consumption and the Air re-breathed*, p. 40.

calculated that in Manchester people die of these complaints at more than three times the rate that people do in breezy Westmoreland. Mere exposure to cold and hardship and privation are not, therefore, to be reckoned amongst the causes of consumption.

But a closer inquiry into the figures laid before us gives not only negative but positive information as to the secondary causes of consumption, and we may safely draw the following conclusions from them.

1. That the occupations of a people have an important influence over the disease.

2. That its fatality increases with the herding together of populations.

It is probable that these two propositions have a good deal in common in respect to the causation of the disease, but it will be convenient to consider them separately and in detail.

1. *Influence of occupation.*—There can be little doubt as to the indications given by our figures, respecting the injurious influence of certain occupations in predisposing to lung-disease, and ultimately to consumption. The mere fact that in most countries where manufacturing processes are carried on the worst places are seen to be the manufacturing centres, would in itself be sufficient to arouse suspicion on this head. Thus, in England, the manufacturing counties of the north-west and west are the highest in the scale of mortality. In Dr. Haviland's map, if we leave out Wales, with its very doubtful statistics, we shall find the spots of darkest blue in the immediate neighbourhood of the industrial centres in Lancashire, Cheshire, and Yorkshire.

In our own chart (see p. 33) the two highest columns of disease are those of Brunn in Moravia and Remscheid in Austria, and both of these are actively engaged in

manufacture, the former of woollen goods, the latter of iron and steel. Nor are we left without direct testimony on this point. Drs. Headlam Greenhow and Arlidge in England, Finkelnburg and Schweig in Germany, Meynne in Belgium, Boudin and Chatin in France—all these observers concur in attributing a malefic influence to certain kinds of work.

In his report to the Privy Council in 1858, Dr. Headlam Greenhow pointed out the influence of occupation as a cause of pulmonary disease; and in 1860 and 1861 he followed it up by two admirable special reports upon the subject.

Without going into the minutiæ of these reports, it may be sufficient to summarise his conclusions, as to the conditions that render workpeople peculiarly liable to these diseases, by arranging them under the four principal heads.

1. The inhalation of dusts of various kinds.
2. Stooping postures at work.
3. Exposure to changes of temperature.
4. Bad ventilation.

But before considering his figures, it is important to observe that in his first report Dr. Greenhow grouped together all forms of disease of the respiratory organs—doubtless in order to avoid errors of diagnosis—and although in his later papers he gives the figures for phthisis and the other pulmonary diseases separately, it is significant of hesitation to accept the figures, that, in his final summing up at the end of each report, he reverts to his former nomenclature, and speaks in general terms of 'pulmonary disease.'

In considering the ætiology of phthisis we are bound to follow the example of this caution, and to decline to accept the figures placed before us as relating to tuber-

cular disease. It is also needful to attempt to distinguish between the direct effects of the occupation itself and those of the ordinary modes of life of the worker.

The subject further opens up wide pathological questions as to the modes in which the injuries sustained by the workpeople lead to true tubercular disease. The discussion of this last-named point would, however, lead us away from the domain of statistics, with which we are now occupied, and it will therefore be best to reserve it for future consideration.

With these reservations we may turn to the conditions mentioned as causes of pulmonary disease:

1. *The inhalation of dusts of various kinds.*—Dr. Greenhow's statistics point to this occurrence as directly productive of pulmonary disease, including phthisis; but we can hardly admit into the latter category many of the cases included under this head in his figures—unless the word is so spoiled as to make it cover those of a non-tuberculous character.

It is certain that the kind of disorder at first produced is not true phthisis. In many cases—such, for instance, as 'grinder's asthma'—the disease is at first one arising simply from mechanical irritation of the air-passages; and this irritation may lead on to emphysema, or to hardening and contraction of the lung-tissues, and ultimately to their destruction, in a way difficult to distinguish from true consumption. In other cases the fine particles of dust of various kinds may even penetrate into the tissues of the lungs and to the bronchial glands, as was shown by Traube, Virchow, and Zenker, and experimentally by Von Ins, Sikorski, and others.

Various forms of these pneumono-konioses have been described, arising from particles of coal-dust, steel filings, dust from stone, glass, or porcelain, and even from

tobacco and cotton-flue. Doubt has been expressed as to whether most of these cases are not truly tuberculous, (notably by Hilton Fagge), but those who have had much to do with such patients cannot share this opinion. I am quite sure that there are many cases of true cirrhosis or fibroid disease of the lung, sometimes produced by irritating dusts, at others by pleuritic attacks often repeated, and ending in thickening and contraction of the interlobular septa, and, so to speak, in a strangling of certain portions of the lung. I have watched such cases for years, and have satisfied myself by repeated stainings that there were no bacilli in the sputum—and after death no signs of tubercle in the lungs. Such cases are, however, with difficulty distinguished from true phthisis. We must, therefore, look with some suspicion upon the tables laid before us; but, inasmuch as tubercle may undoubtedly be engrafted upon a lung injured by dust, it behoves us to inquire into the relative proportions of persons engaged in different trades, who are said to have died of phthisis.

The observation as to the injurious influence of dusts in this regard is by no means a new one. I need only note the elaborate works of Ramazzini and Thackrah on the subject.

Wepfer, in 1727, speaking of millstone-grinders, says, 'omnes phthisici fiunt.'

Even if we assume that many workpeople commence with simple irritative diseases, we cannot refuse to recognise their trades as truly causes of consumption, if we find that large proportions of those who work at them ultimately fall victims to that malady.

All dusts, however, are not equally hurtful. The following lists, abridged from Hirt's tables, show how greatly the incidence of phthisis from this cause may vary.

TABLE V.—*Percentage of Phthisis in total numbers of Sick amongst Workmen exposed to the Inhalation of Dusts of Various Kinds*

Metallic		Mineral	
Needle-polishers	69·6	Flint-workers	80·0
File-cutters	62·2	Grindstone-makers	40·0
Lithographers	48·5	Stone-cutters	36·4
Grinders	40·4	Plasterers	19·0
Dyers	25·0	Porcelain-makers	16·0
Painters	24·5	Potters	14·7
Printers	21·6	Masons	12·9
Tinkers	14·1	Cement-makers	8·0
Vegetable		Animal	
Cigar-makers	36·9	Brushmakers	49·1
Weavers	25·0	Hair-workers	32·1
Millers	10·9	Turners	16·
Bakers	7·0	Button-makers	15·0
Mixed		No dust	
Glass-cutters	35·0	Shoemakers	18·
Glass-makers	17·8	Glovers	10·0
Day labourers	15·1	Butchers	7·0

It is evident from this list that the sharpest and most irritating dusts are those that tend most surely to produce lung-disease, ending eventually in consumption.

The hardest and most pointed dusts, such as those from steel and flint, produce the most baneful effects, and after them come fine cuttings from bristles and the spiculæ of glass and porcelain.[1] The dust of flour and the delicate fibres of cotton and wool do but little harm. This result is in accordance with our experience of the influence of the staple manufacture of my own neighbourhood. In an inquiry as to the health of cotton operatives, Mr. Royston and I discovered that these workpeople are not more liable to consumption than the ordinary labourers of the town, the percentage of deaths

[1] This observation coincides with the results of Dr. B. W. Richardson's inquiry.

from affections of the lungs being 61 for the former and 60 for the latter.[1] We came to the conclusion that, 'although these affections may be partly due to mechanical irritation from cotton-fibre and other substances arising from the work, they are still more frequently produced by the subtle diffusion of smoke in the atmosphere and the general vitiation of the air by noxious vapours and gases.' Dr. Tracy, in Buck's 'Hygiene,' says, 'It is doubtful if cases of phthisie cotonneuse occur at all in this country.'

There are some differences between the arrangement of the trades in the order of their degrees of danger, as given by Hirt and by others; thus Eulenberg, in the 'Handbuch der Gesundheitswesens,' with regard to the mortality in Prague, gives the order of phthisis-mortality as follows: bookbinders, goldsmiths, glove-makers, stone-cutters, printers, turners, waiters, forgemen, locksmiths, bakers, joiners, potters, barbers, coopers, musicians, braziers, shoemakers, varnishers, tailors, furriers, weavers, masons, butchers, brewers, tanners, mountaineers, gardeners, millers, room-decorators, shipbuilders, coachmen.

The differences between the several sets of statistics are probably due to variations in the conditions under which the several classes of artisans did their work, but they add to our reluctance to receive the figures as evidence of the influence of the trades themselves, and make it probable that the conditions in question had at least as much to do with the result as the occupations.

In this regard, it is interesting to note that Seltmann[2] and Mirkel believe that the inhalation of carbon is antagonistic to the development of tubercle, and Mr.

[1] 'Transactions of the Social Science Association,' 1867, p. 467.

[2] *Deutsches Archiv*, vol. ii.

Simon [1] has called attention to the fact that the coal-miners of Durham and Northumberland are not especially liable to lung-disease, and he accounts for this by pointing out the good ventilation of the mines in which these men work.

2. *Stooping and constrained postures during work.*—The next great exciting cause of phthisis mentioned by Dr. Greenhow is the interference with the respiratory apparatus by posture, and he adduces as evidence the heavy mortality of the women and girls employed in the lace and hosiery finishing and winding looms at Nottingham, the watchmakers of Coventry, the handloom weavers of Macclesfield and Leek.

The following table, drawn from Dr. Greenhow's reports, shows how greatly the phthisis-ratio of these artisans exceeds the standard.

TABLE VI.—*Death-rate per* 1,000 *living from Pulmonary Affections*

	Males	Females
Nottingham	8·13	7·03
Coventry.	6·61	5·73
Macclesfield	7·43	8·51
Leek	7·80	8·51
Six northern standard districts . .	2·97	3·04

It is again difficult to separate the operation of this condition of work from other influences adverse to health, such as insufficient food and bad air.

Still it is evident that, from some cause or other, workpeople, who have to carry on their occupation in constrained postures, do actually suffer more than others from consumption, and we shall presently see that modern research affords a reason why this result should probably follow.

[1] *Fourth Report to the Privy Council.*

On the other hand, it is only right to notice the observation of Rokitansky, Wagner, and others, and which has been generally confirmed by experience, that hunchbacks are but seldom affected by tubercular disease of the lungs. (I can myself only remember three such cases in my own practice.)

Moreover, the miners, whose exemption from phthisis has already been mentioned, work in the same cramped postures as their brethren in other coalfields.

3. *Exposure to variations of temperature, moisture, &c.*—This includes the third class of conditions that, according to Dr. Greenhow, render workpeople peculiarly liable to pulmonary disease; and he especially instances the cases of slip-makers in potteries, and the spinners of flax, who are naturally exposed to heated and moist atmosphere, and the workpeople in overheated factory rooms.

There can be little doubt that these conditions, taken in connection with bad ventilation, have much to do with the prevalence of diseases of the respiratory organs; but, as we have already seen, they can only be very remotely implicated in the causation of phthisis, and I shall not therefore dwell further on this point. I have already shown that exposure to the weather alone is actually a preservative against both catarrhal affections and phthisis, and the statistics of occupations abundantly confirm this view. Engine-drivers and stokers on railways are certainly not specially liable to these disorders. Blacksmiths and charcoal-burners are peculiarly free from them. Forgemen, iron-puddlers, and glass-blowers also are not prone to contract consumption, though the last-named class are subject to emphysema.

The stokers on steamships must indeed be excluded from the category of persons thus free from a consump-

tive tendency; but another cause affects them, for their quarters are, for the most part, very contracted and badly ventilated.

4. *Working in ill-ventilated rooms.*—We have now reached the last of the conditions mentioned by Dr. Greenhow, as tending to the production of consumption amongst workpeople, and it is impossible to resist the conclusion that it is the most powerful of them all. Most, if not all, of the conditions that we have found to lead to consumption have had associated with them air rendered impure by breathing, and allowed to stagnate through want of proper ventilation. The dusty atmosphere in workshops is also often a confined atmosphere; the persons who work in a constrained or stooping posture do so for the most part in crowded, ill-ventilated shops or workrooms, or in the recesses of mining galleries; and the effects of variations of temperature in factories are aggravated by the same cause. But there is plenty of direct evidence on the point. Dr. Greenhow's inquiry, in 1860, led him to the conclusion that 'in proportion as the male and female populations are severally attracted to indoor branches of industry, in such proportion, other things being equal, are their respective death-rates by lung-disease increased.

He instances especially as occupations injurious in this regard:—1. The making of earthenware and china, as at Stoke-on-Trent and Wolstanton; 2. Tin and copper mining (Penzance and Redruth); 3. Lead-mining (Reeth); 4. Flax factories (Pateley Bridge); 5. Silk-working (Macclesfield and Leek); 6. Wool factories (Leeds, Bradford, &c.); 7. The making of hosiery (Leicester and Hinckley); 8. Cotton factories (Preston); 9. Lace-making (Towcester and Newport-Pagnel); 10. Straw-plaiting, (Berkhampstead); 11. Glove-making (Yeovil).

This is a long list, and it already contains several of the trades whose tendency to phthisis had been set down mainly to other causes, notably, the potters of Stoke-on-Trent, whose illness had been ascribed to dust; the silk-workers of Macclesfield and Leek, who were supposed to have suffered from stooping during their work; and the spinners of flax, who are also exposed to variations of temperature; but, in truth, Dr. Greenhow's reports abound in notices of the bad ventilation of all kinds of workrooms, and instances of the good effect of ample cubic space, and of free currents of air.

Thus he mentions the intolerance of ventilation shown by mould-makers at Stoke,[1] the bad ventilation of flax factories at Pateley Bridge (p. 149); the overcrowding and want of ventilation of living rooms and factories at Macclesfield and Leek (p. 155). In one of the latter the space was less than 200 cubic feet per head, and of ventilation there was none.

But, perhaps, the most striking proof of the influence of confined air, in the production of consumption, is to be found in the relative death-rates from this disease amongst males and females respectively, which have already been given. (See Table IV. and Diagram, p. 39.) The greatest range of difference in these rates is found in the agricultural districts, where the men are mostly employed out of doors, and are but slightly affected, whilst the women are employed at home, and die of consumption at twice the rate of the men. I would especially instance Battle, Pickering, and Billesden. It will further be found that in manufacturing towns, the incidence of phthisis is heaviest upon that sex which has most to do with indoor employment.

On the whole, as Mr. Simon says,[2] in his summing

[1] 3rd Report to Privy Council, p. 10. [2] 4th Report, p. 14.

up of the evidence, 'among the aggravating circumstances that may indefinitely increase this evil (lung-disease), probably none is so effective as the bad ventilation of the work-place.'

'The concurrent testimony of these most various illustrations is—that, commonly, where many persons are employed together at any indoor industry, the ventilation of the work-place is likely to be so bad as to convert the employment, which perhaps is not in its own nature of hurtful tendency, into an employment seriously dangerous to health. Here lies the explanation of a fact most deplorable for the working classes of our country—that, in proportion as the people of a district are attracted to any collective indoor occupation, in such proportion, other things being equal, the district death-rate by lung-diseases will be increased. For the bad ventilation, which, as a rule, belongs to the place of employment, tends to develop among the workpeople a large excess of phthisis, and probably also some excess of other fatal lung-disease.'

5. *Air rendered impure by respiration.*—We are thus led inevitably, by the statistics, to the reason for Dr. Greenhow's second proposition, that the fatality of lung-disease increases with the herding together of populations in large towns, and this is, that the truly infective impurity of the air must be in some sense a respiratory impurity. We have already seen that the specific organism of tubercle has been discovered in the air of consumption hospital wards—in the dried-up sputa of phthisical patients, and even, though rarely, in the aqueous vapour exhaled from their lungs. It was therefore certain that the disease must be mainly derived from this source; but we now see, from the concurrent testimony of facts, drawn from the geographical distribution of the disease, and from the rates of mortality from lung-

disease amongst workpeople, that the power for evil of the disease-germ increases, *pari passu*, with increase in the density of the poison.

Baudelocque, in his treatise on scrofulous disease, was perhaps the first clearly to see that this was so. Speaking of air rendered foul by breathing, he says: 'This is the true cause—perhaps the sole cause—of the disease of scrofula,' and he adduces many proofs in support of his theory.

Sir James Clarke also, in 1835, in his monograph upon consumption, speaks of 'the respiration of a deteriorated atmosphere as one of the most powerful causes of the disease.' [1]

In one of his early letters to the Registrar-General, Dr. Farr gave some statistical evidence to show that the mortality from diseases of the respiratory organs and from phthisis is in direct ratio to the density of population. The following table is drawn from the annual report.

TABLE VII.—*Mean Mortality in three groups of the Thirty-two Metropolitan Districts*, 1839

Districts	Square yards to one person	Annual rate per 100,000		
		Totals	Respiratory organs	Phthisis
1 to 10	57	3321	822	478
11 „ 20	78	2839	768	451
21 „ 30	217	2169	588	354

The table (p. 5) showing the mortality in different parts of the world, drawn up for the Army Sanitary

[1] But the truth was hardly fully accepted until Dr. McCormac took up the subject, and with iteration insisted that, 'wherever there is foul air, unrenewed air, impure air . . . . there we meet consumption, there we meet scrofula and an untimely death.'

Commission, bears witness to the same facts; and the commissioners, in their report, point out that 'in civil life, insufficient clothing, insufficient and unwholesome food, sedentary and unwholesome occupations, and the vitiated atmosphere of unhealthy dwellings, all contribute to the propagation of this class of diseases. But in the army it cannot be alleged that the clothing, the food, or the nature of the occupation in itself are of a character which would justify the imputation that they are among the predisposing causes of the excessive mortality of the soldier by pulmonary disease.

'The ravages committed in the ranks of the army by pulmonary disease are to be traced in a great degree to the vitiated atmosphere generated by overcrowding and deficient ventilation, and the absence of proper sewerage of barracks.'

In reference to this Dr. Farr said: 'The prevalence of phthisis in the armies of Europe is probably due in part to the inhalation of expectorated tubercular matter, dried, broken up into dust, and floating in the air of close barracks,' and Dr. Parkes remarks that this prevalence 'can scarcely be accounted for in any other way than by supposing the vitiated air of the barrack-room to be chiefly at fault.' Mr. Welch, of the Army Medical School, Netley, also proves that consumption in the army increases with length of service, and that it is due in the first place to 'vitiated barrack atmosphere,' and to constant irritation of foul air inspiration . . . the chief deleterious agent in the generation of consumption being the organic matter, which, taken into the air-passages, there lodges, and chronically irritates.' [1]

[1] Prize Essay on *The Nature and Varieties of Destructive Lung Disease, as seen amongst Soldiers.* See also a paper by Robert Lawson, Esq., 1st Milroy Lecturer, in 'Trans. Statistical Soc.,' vol. i. p. 470.

Dr. Parkes, in his work on hygiene, gives numerous instances of the influence of re-breathed air in the production of phthisis—instances drawn from the history of prisons, gaols, and workhouses ; and Hirsch, in his articles on scrofula,[1] and on consumption,[2] gives long lists of examples of the origin of both diseases under circumstances in which confined air must have been the principal factor.

But we have still not quite reached the limit of the aid to our inquiry that may be obtained from statistics of disease. We may push our investigation into still closer quarters, and, abandoning total death-rates of institutions and towns, we may examine into the mortality of certain districts, and even of certain houses.

In a paper on 'Tubercular Infective Areas,' read two years ago before the Epidemiological Society, I gave the details of an inquiry made into the incidence of phthisis in some of the worst districts of Manchester and Salford—an inquiry that was greatly facilitated by the kindness of the Medical Officers of Health of these boroughs, and by the excellence of the mortality tables which they placed at my disposal. Its results showed that the portions of these districts most affected by the disease were the close courts and alleys, the shut-in or blocked-up lanes, and above all the houses built back-to-back with no thorough ventilation. I especially noted the cases in which, in the space of five or six years included in the inquiry, double or treble occurrence of the disease had taken place in the same houses, and I found them very numerous.

These results have been confirmed by other observers. Thus Dr. Niven, of Oldham, writes to me respecting 3,001 deaths from tuberculosis, which occurred in that town

[1] *Handbook*, ii. p. 632. [2] Vol. iii. p. 222.

during eleven years (1877 to 1887), and states that they took place in the worst class of houses, and in 302 cases there were two or more in a house. He has calculated mathematically the chance of any one house being twice affected (not infectively), and he finds that only sixty-eight, on this hypothesis, would have thus suffered, whereas 274 houses were so attacked, and that whilst not more than 7·6 houses should have suffered thrice, the actual number so attacked was 24.

In a joint report to the Local Government Board, made in 1888, by Dr. Barry and Mr. P. Gordon Smith, on 'Back-to-Back Houses,' which included several of the districts which I had selected, the conclusion formed upon this point is, 'that it appears probable that the want of through ventilation . . . gives rise to an increased mortality from pulmonary disease, phthisis, and diarrhœa.'

Again, Dr. Flick, of Philadelphia, has recently carried out an elaborate topographic study of phthisis in that city, extending over a period of twenty-five years, and he draws the following conclusions:—(1) 'that a house which has had one case of consumption will probably have another within a few years, and may have a very large number of cases in close succession;' (2) 'that when a case of consumption occurs in a house, approximate houses are considerably exposed to contagion;' (3) 'that houses in *localities* where endemic after endemic has existed, have nevertheless escaped the disease;' (4) 'that tuberculoses of different kinds occur in the same localities, and often on the same lots as consumption,' and (5) 'that during the twenty-five years scarcely 20 per cent. of the houses of the ward were so affected.'

He ascribes these results to contagion in the houses themselves.

These facts must be placed in apposition with Dr. Cornet's researches upon the bacillus-holding properties of the walls of houses in which consumptive patients have resided. They may probably afford an explanation of many of the facts detailed in the course of this lecture.

In the next lecture I will return to the subject of contagion.

LECTURE III

PREDISPOSING CAUSES OF PHTHISIS

Universal distribution of the bacillus—Numbers ejected by consumptives —(*A*) Reasons for immunity—Mechanical and physiological—Dosage of bacilli—Varying susceptibility—Mitral stenosis—Lithiasis—Copper and wool workers. (*B*) Predisposition to infection—The tuberculous constitution—Hereditary tendencies—Direct infection—Latency—Vulnerability—Age—Sex—Race—Influence of previous disease. (*C*) Modes of infection: by the skin, generative organs, the digestive tract, the lungs—Food—Flesh of tuberculous animals —Milk—The atmosphere—Direct contagion—Sputum—Dust—Infected houses—Convents. (*D*) Conditions enhancing infectiveness of bacillus—Foul air, dampness of soil, organic vapours.

The considerations and the evidence adduced in the preceding lectures may perhaps be sufficient to show the part that is played by the bacillus of tubercle in the production of the lesions of phthisis, and to indicate the chief circumstances that influence its general distribution amongst groups of mankind.

We shall to-day have to consider both the conditions that render individuals liable to contract the disease, and some of the modes in which the parasite gains an entrance into the body.

We have already seen that the disease may be found everywhere that men are congregated together, or, as it might perhaps be more correctly stated, everywhere that tuberculous individuals are, or have been, present within a recent period. Nor is it difficult to understand why this should be so, in the light of modern research. It

has been calculated by Bollinger that one phthisical person may eject from his body in the course of twenty-four hours no fewer than 20 millions of bacilli. Moreover, for the greater part of their illness, consumptives are not confined to their rooms or to their houses. They go about like other people, and mix in crowded assemblies, discharging their bacillus-laden expectoration in every convenient place. The sputum thus ejected has been found by Koch to possess a marvellous tenacity of life, being still virulent in the dried-up sputum after a lapse of several months.

The walls of houses, and the dust that flies about in the air, have been shown by Cornet to contain the virus in an active condition.[1]

It is certain, therefore, that the germs of the disease are almost everywhere present, and that we all of us inhale some of them in the course of each day or week.

It would seem also at first sight that, once received within the body, the bacillus would have no difficulty in obtaining nourishment and in growing. Professor Koch's observations show that the bacillus requires only a certain amount of moisture, a temperature of between 82° and 107° Fahr., and a supply of nitrogenous food, such as blood-serum—all of which conditions are to be found within the human body. The presence of phthisis in our midst ought not then to be difficult of explanation; we should rather, in the first place, seek out the reasons for the exemption of so many from the results of infection.

REASONS FOR IMMUNITY FROM TUBERCULOSIS

Amongst the conditions necessary for the growth of the bacillus, there is yet one that was mentioned in the lecture, and which is of great significance, namely, that it requires

[1] *Berliner Med. Woch.*, 25 März, 1889.

a sojourn of one or two weeks, and sometimes more, in a suitable medium before it can take root, so to speak, and develop into a colony.

Now from Watson-Cheyne and Dr. Kidd and others, we learn that the epithelium of the ultimate portions of the air-passages is usually the first point of attack by the organism. We may therefore suppose that it is necessary for the organism to effect a lodgment somewhere in this position.

Or again, it may perhaps make its way into the inter-alveolar lymphatics by means of the prolongations of the branched cells of the alveolar septa, called by Dr. Klein 'pseudostomata,' which he has shown to be capable of taking up foreign bodies, such as oxide of iron, silica, and even carmine (Sikorski).

It can hardly be doubted that, if these inorganic particles can thus find an entrance, the bacillus of tubercle may do the same, and thus the way into the body might be supposed to be opened to the organism; and, once planted in such soil, if it can remain there a sufficient length of time, it will find a suitable temperature and ample supplies of the nourishment that it requires; but there are in the healthy body various impediments, mechanical and physiological, which it is difficult for it to over-pass, and which will probably account for the immunity of all those in whom these barriers have not been previously broken down.

1. We have the difficulty with which minute bodies such as the bacilli could reach the ultimate air-passages.

It is true, as Professor Tyndall has shown by means of his illuminated tube, that the lungs possess a sort of filtering action, catching up the organic dust inhaled with the air, and preventing it from coming forth again with the returning breath, but this action is due to the en-

tangling of the dust by the mucus lining the air-passages. Before it has reached the ultimate ramifications of these tubes it is caught on the glairy mucus, and is speedily ejected by the action of the ciliary waves.

That this filtering action is not complete, however, I have myself proved, both by the microscopical examination of the aqueous vapour of the breath, in which there were numerous epithelial scales and other objects, and by the detection of a few, very sparsely scattered, bacilli in the breath of certain consumptive patients.

In some forms of lung-disease, also accompanied by copious bronchial secretion, the probability of this event is increased, especially if the accompanying cough prevents the lodgment of mucus in any of the air-tubes. As has been shown in the research just mentioned, the organic matter of the breath is always less when there is much secretion. The proportionate amount of this substance exhaled in the aqueous vapour from the lungs, was only one-half of that from healthy persons, not that there is really less organic matter thus excreted, but because it was taken up by the mucus before it could reach the mouth. The immunity from phthisis of many cases of chronic bronchitis and emphysema is, perhaps, due to this cause. On the other hand, the influence of a lessened mobility of the chest, and the consequent partial stagnation of the air, is shown in the tendency of the upper lobes of the lungs to tubercular deposit, and we may place in the same order of predisposing causes, the evil results of stooping or constrained postures during work. Perhaps, too, the well-known tendency to tubercular infiltration of parts of the lung confined by pleuritic adhesions, or consolidated by catarrhal pneumonia, may be due to the facility afforded for a prolonged lodgment of the bacillus, and the same may be said of hæmorrhagic

deposits in the lungs. In this case the organism would receive both board and lodging for a term sufficient, in many cases, for its development.

2. Another barrier to infection from the bacillus, even if it obtains a temporary lodgment, may be found in the vital processes carried on in the lungs. I am not aware of any direct experiments upon the action of nascent oxygen, or of ozone, upon the virulence of the bacillus; but my own experiments with the latter agent in fifteen cases of phthisis seem to show that it has a distinct influence in enabling the system to tolerate the presence of these organisms, and gain health and strength in spite of their continuance in the body. Moreover, the abundant records of the good effects of open-air life, and of free ventilation, seem to show that the disease cannot gain ground under these conditions. It seems highly probable that the bacillus of tubercle, though it may not be destroyed at once by these agents, yet is robbed of its power for evil by the same processes that Dr. Angus Smith and Pasteur have proved to take place in certain other organised ferments.

In any case, whether by simple oxidation, or by the energy of other vital actions—probably also, as Koch and others have shown, by the direct antagonism of phagocyte corpuscles—it is certain that the healthy human body has the power of disposing of a certain quantity of these creatures, even after they have been directly injected into the system.

3. There remains the question of dosage. Many of the facts ascertained, with regard to the spread of ordinary epidemic infectious diseases, seem to show that a certain concentration of the virus is necessary, before they can overcome the resistance opposed to them by the bulk of the population. The definite cyclical periods, observed

by many of these disorders, render it highly probable that a certain density of the susceptible population must be attained, before they can spread through large communities (Epidemic Cycles, 'Proc. of Lit. and Phil. Society, Manchester').

The recent observations of Bollinger on this point, with reference to tubercle,[1] are of great interest.

He shows that very dilute solutions of sputum (1 in 100,000) and of cultures of bacilli (1 in 400,000) are virulent when injected into the peritoneum, but that much lesser dilutions were inoperative upon the intestine. In these researches the peritoneum remained sound in two-thirds of the cases, but the disease attacked the lymph-glands, the spleen, and the lungs. Incidentally it may be remarked that this observation shows that lung-tuberculosis need not always depend upon inhalation, but may arise from virus that has been swallowed or injected.

But in addition to these evidences of varying susceptibility to the poison, Dr. Bollinger ascertained that whilst 800 bacilli, when injected into the peritoneum, are sufficient to cause the disease, smaller numbers than these become less and less certain in their operation.

We may gather from these facts the lesson, that up to a certain point the healthy body is able to destroy, or at least render innocuous, the organisms that gain access into its interior. There are probably great differences in this respect in different individuals, just as we know that some people seem to be quite proof against the infection of certain exanthemata, whilst they are especially prone to others.

It is worth noting that certain diseases, such as

[1] Record in the *Deutsche med. Wochenschrift* for October 1889, p. 849.

mitral stenosis and gout or lithiasis are supposed to confer an immunity to the disease, but the explanation of this phenomenon is not yet clear.[1] The workers in copper are said to be more exempt than other people from phthisis, and Sir J. Simpson thought that the woollen manufacture conferred a certain degree of immunity upon the mill-hands of Yorkshire. These are some of the reasons for immunity.

REASONS FOR INFECTION

It behoves us next to inquire whether there are any characteristics that make men unusually liable to contract tubercular disease.

It has long been assumed that there is a scrofulous diathesis or tuberculous constitution, and in 1860 Sir W. Jenner gave his classical description of the contrast between these two types of temperament. Since Galen also it has been supposed, that a peculiar form of configuration of the chest predisposed persons to contract the disease, a supposition advocated by Traube in Germany and by Dr. Gee in England.

I do not know how far these views are accepted at the present day. It seems highly probable that a certain form of chest, with insufficient expansive and expulsive power, may afford opportunities for the deposit of the tubercle-bacillus. But, on the other hand, I have seen men of the finest physique, and with magnificently developed chests, fall victims to the disease. I apprehend that many of the cases recurring amongst Her Majesty's foot-guards must have been of this description.

[1] Verneuil has only found two cases of phthisis associated with stone in the bladder, and finds upon inquiry that it is altogether exceptional (*Etudes*, i. 229). E. Solles (*Etudes*, ii. 95) regards erysipelas as also ntagonistic.

I. HEREDITARY TENDENCIES

Few medical men who have been long in practice will doubt the existence of family predisposition to tubercular disease. Thus most of us have seen instances of families in which almost every member has died of the disease, and others in which members of the same family, living in different and sometimes far distant places, have yet most of them ultimately succumbed to it. Yet it is quite possible to make too much of this influence.

When we consult the statistics that have been put together on the subject, we find great differences in the results given by different observers.

Thus Briquet found that one-third of the consumptive patients at a hospital were born of consumptive parents on one side or the other; Dr. Quain, 25 per cent.; Dr. J. Pollock, 30 per cent.; Dr. C. T. Williams, 12 per cent. of direct influence, and 48 per cent. of family predisposition. Rilliet and Barthez found that one in seven of tuberculous children had some hereditary taint; Mr. Phillips only 4 per cent., Lebert three-fifths. Lugol states that more than half the subjects of scrofula have consumptive progenitors. Francis Galton[1] gives 26 per cent. of consumptive children by one method of calculation, and 28 by another. But in these figures no account is taken of the influence of external circumstances—sources of infection from without that are common to all the members of the family.

Again, there are so many deaths from phthisis in the country—nearly half of all the deaths between the marriageable ages, from fifteen to thirty-five, are due to this cause—and hence without any such thing as

[1] *Natural Inheritance*, p. 173.

hereditary taint, there would be nothing surprising in the fact that half of the consumptive patients have had consumptive relatives, unless the families were unusually large, and if we draw grandparents and collateral relatives into the statistical net, it breaks at once and holds no solid conclusion.

Dr. Walshe (the chief authority on chest diseases in this country) obtained from his hospital patients the result that about 26 per cent. came of father or mother, or of both parents similarly diseased; but in discussing the significance of these figures, he asks whether they prove the reality of hereditary influence, and decides that they do not. 'This ratio,' he says 'of 26 per cent. might be, and probably is, no higher than that of the tuberculised portion of the population generally,' and he concludes that, 'much phthisis is, in each generation, non-hereditary.'

It is, moreover, highly probable that heredity has much less to do with consumption than is commonly supposed. A very large proportion of cases arise without any phthisical family history of the past. Many healthy families leaving the country and coming to reside in crowded towns, lose some members subsequently from consumption. In the army more than 60 per cent. of cases are non-hereditary.

It will thus be seen that, although we cannot say with Louis,[1] 'Nous n'avons recueilli aucun fait en faveur de l'hérédité de la phthisie,' yet we must ascribe to it a much smaller influence than is usually given.

Granting also the real influence of heredity in predisposing to tubercle, there are at least three distinct modes in which it may be exerted. 1. By the direct infection of a child with the disease in an active state.

[1] *Recherches sur la Phthisie*, p. 532.

2. By the implantation of the organism or of its spores, which may remain for a period latent in the system. 3. By the transmission of a constitution peculiarly open to attack by the bacillus—in other words, an undue vulnerability of the system.

It is possible that each of these circumstances may occur. 1. We occasionally see cases of undoubted congenital tuberculosis, and the researches of Landouzy and Martin, of Lannelongue and of Niepce,[1] show clearly the possibility of this event.

The first-named authors show (pp. 59–74) the tuberculising power of the seminal fluid of tuberculous guinea-pigs. Niepce found bacilli in the semen of tuberculous individuals, and Lannelongue declares in favour of the doctrine of intra-uterine infection of the fœtus (p. 97), citing in support the aforesaid researches, and the observations of Demme, who found in two infants, one of whom died on the twenty-first day after birth, an intestinal tuberculosis, and the other at the twenty-ninth day, a pulmonary tuberculosis with an advanced cavity in the lung. He opposes these facts to the assertions of Leudet, who had never seen a case of fœtal or congenital tuberculosis, whether hereditary or acquired; of Vallin, who speaks of it as so rare as not to be worth notice; and of Professor Peters, who declares roundly that 'on ne naît pas tuberculeux mais tuberculisable' (p. 94). Bollinger[2] says, 'Die sogenannte congenitale Tuberculose so gut wie auszuschliessen ist.'[3]

2. Lannelongue ascribes the rarity of early tuber-

[1] Verneuil, *Etudes sur la Tuberculose.*

[2] *Münchener med. Wochenschrift*, 17. Juli, 1888, p. 486.

[3] An interesting fact in relation to this point is given by M. Duprey, that in a flock of merinos, a phthisical ram produced from sixteen to twenty tuberculous sheep (*Traité de l'Affection Tuberculeuse chez les Animaux.* Paris, 1817).

culous infection to the conditions of resistance in the bodies of infants and children, which may have the power of keeping the virus in a *latent* state, and which may even prevent its development altogether.

Baumgarten[1] goes much farther than this, and boldly says that, 'Tuberculosis may remain latent, under certain circumstances, during the whole of life, without interfering with living functions in any observable degree.' Nay more, he believes that it may pass through a generation and be transferred from grandparents to grandchildren. He places it in the same category with syphilis and leprosy, and ascribes the greater part of the tuberculosis in the world to this hereditary descent.

He defends this thesis by an appeal to the experiments already quoted of Landouzy and Martin, Queyrat and Lannelongue,[2] and cites in its favour not only his own researches but those of Marchand,[3] of Bollinger,[4] and other pathologists, though, as we have seen, Bollinger is certainly not on his side. He also endeavours to show the inadequacy of other explanations, such as infection by the air, by food, &c.

But, with all respect to so able a pathologist, I must say that Professor Baumgarten has failed to convince me of the truth of his theory.

There is no direct proof that the virulence of the bacillus can remain latent for more than a few months.

[1] *Lehrbuch der pathologischen Mykologie*, p. 631.

[2] 'Die neuen Anschauung über die Natur der Tuberculose,' *Deutsche med. Woch.* 1883, No. 15.

[3] 'Ueber Entstehung und Heilbarkeit der Tuberculose,' *Münchener med. Woch.* 1888, 29, 30.

[4] Gosselin (*Etudes sur la Tuberculose*, Verneuil, p. 34) also gives instances in which iodoform seemed to render sterile the thousands of bacilli injected into rabbits; but he remarks that it seems impossible that they can remain inactive for several months and then revive.

The heredity of leprosy is still unproved, and even if the evidence in its favour were stronger, the nature of the hereditary tendency to contract it is as uncertain as is that to tuberculosis.

The analogy of syphilis with tuberculosis is also not yet made out, and the character of its latency is still *sub judice*. Dr. Baumgarten also surely under-estimates the great weight of the evidence that connects phthisis with the inhalation of impure air, and with indoor occupations. On these grounds, therefore, we certainly ought to hesitate before accepting the fatalistic doctrine of the inevitable nature of inherited tuberculosis.

3. The last-named form of hereditary influence is probably the most common.

We know that a tendency to contract other diseases than phthisis is not unfrequently transmitted from parents to children. Our own royal family have shown a peculiar proclivity to both typhoid fever and diphtheria, and a similar predisposition to contract these diseases occurs in other families. I know, for instance, of one family in which six out of eleven children have died of diphtheria, and other members of the family have suffered from, but have survived, this complaint. They were not all of them struck down at the same time, or by the same epidemic; but three children died in South America, one in the South of England, and two in the place where they are now residing. Such a fatality as this from a particular disease displays a strong family predisposition both to contract the disease, and to succumb to its attack.

Much the same must be said of a hereditary tendency to consumption; but there is no more need to assume the existence of a tubercular constitution than of a diphtheritic or typhoid diathesis.

Villemin distinctly repels the idea of the existence of such a constitution, and says of heredity that 'the sole influence it exerts is in the transmission of an aptitude to contract the disease.'

Niemeyer says, 'I do not hesitate to say, in spite of all assertions to the contrary, that it is by no means proved that tuberculosis, in the strictest sense, is an inheritable disease;'[1] and again, 'Quite as decidedly as we have opposed the *evidence that tuberculosis is inheritable*, must we pronounce in favour of a frequent occurrence of an *inherited disposition to pulmonary phthisis*' (p. 20).

I admit the existence of hereditary predisposition to tubercle; but I do not attribute to it a great importance; and, if we set aside the few congenital cases of the disease, I believe that the transmission of an aptitude to contract it, does not necessarily lead to the disease itself—if care is taken to avoid the sources of external infection. In short, I am inclined to agree with Louis when he declares that 'few persons are born necessarily to die of consumption.'

II. AGE, SEX, AND RACE

Somewhat similar remarks must be made with respect to the influence of age, sex, and race upon the tendency to contract the disease. We have already seen that, up to the age of puberty, the mortality from phthisis is comparatively small; but after this age the younger residents in an infected house are always the first to succumb to the disease.

This fact comes out in many of the returns obtained by the Collective Investigation Committee of the British Medical Association. In several of the cases of the

[1] *Lectures on Phthisis*, Syd. Soc., p. 19.

almost wholesale destruction of a family residing in one house, the older members, the father and mother, escape the disease.

Sex seems to have only slight influence—it is at any rate as nothing, against the overpowering presence of such circumstances as indoor employment, unhealthy trades, &c.

All races also, as we have seen, suffer more or less from the disease, though it is acknowledged that the negro, as well as the monkey, suffers grievously when transplanted from his own land.[1]

III. THE INFLUENCE OF PREVIOUS DISEASES—EITHER GENERAL DISEASES OF THE BODY, OR LOCAL DISEASES OF THE LUNGS THEMSELVES

It is well known that certain disorders, such as enteric fever and measles and small-pox, especially after an imperfect recovery, often leave behind them a serious tendency to tubercular disease. This tendency may be due simply to their debilitating influence, or more frequently to the fact that by reason of the lesions of surface that have taken place in the course of these diseases, a portal has been opened through which the bacillus of tubercle can enter.

Over-lactation, frequent pregnancies, diabetes, and other debilitating disorders are probably predisposing causes, as rendering the system less able to destroy the organisms that accidentally make their way into the system. But it is to previous affections of the lungs that we must look, as the most common causes of bodily predisposition.

[1] It must be pointed out, however, that in most cases he is lodged amidst most unhealthy conditions, when he is thus brought over.

Most phthisical subjects refer the onset of their disease to a 'chill,' or to a 'neglected cold,' and some observers, such as Dittrich, Niemeyer, and Buhl, went so far as to ascribe nearly all cases of tuberculosis to 'catarrhal pneumonia,' a disease which was supposed to be the main source of the caseating masses found in phthisical lungs after death. In this opinion they were followed by many other pathologists. Recent researches have, indeed, proved the erroneous nature of these conclusions, and have established Laennec's doctrine that these masses are themselves truly the result of tuberculosis, but they have not affected the observation that many cases of phthisis are preceded by purely inflammatory diseases of the lungs, such as pleurisy, acute bronchitis, broncho-pneumonia, and imperfect resolution of ordinary pneumonic inflammation.[1]

It is not difficult to understand why these diseases should prepare the way for the entrance of the tubercular virus. We have seen the nature of the safeguards against infection that exist in healthy lungs; but in

[1] 'In my experience croupous pneumonia is the commonest form which produces a fibroid change. The unabsorbed residue sets up an irritation, which is followed by increase of the normal fibrous tissue until large bands of this are seen in parts of the lungs; the process is a very chronic one, and is generally associated with much pigmentation and anthracosis. In these cases—at any rate in some of them—the consolidated material after a time caseates and breaks down, and in this can be found the tubercle-bacilli. I have noticed one peculiarity in connection with this disease, that is the formation of large giant-cells at the edges of the consolidation, together with fibroid tissue resembling tubercles. This was of quite recent date and probably corresponded to the breaking down of the consolidation. In miner's and other kinds o phthisis, caused by long-continued irritation, the process is essentially the same; it results in the formation of dense fibrous tissue. The breaking down in these cases seems to be independent of the origina disease.' Gibbes and Shurley, 'Etiology of Phthisis,' *American Journ. of Med. Science,* March 1890.

acute bronchitis one of the earliest changes is the detachment of the ciliated epithelium of the affected bronchi, and this remains absent during the course of the disease. It is true that there is also, usually, an abundant proliferation of epithelium and of mucous fluid from the mucous glands, and that this may, and generally does, arrest the progress of infection; but any germs that escape this trap will probably find their further progress facilitated by these changes. Koch ascribes the tuberculosis following measles to this source.

Even in chronic bronchitis, after a time, the expulsive machinery may become defective, the waving cilia may become less active, the muscular apparatus of the tubes may be weakened, and dilatation and plugging of the air-passages may occur; thus the bacillus may find a lodgment within the lungs, and true tubercular disease may be set up. This specific infection is again still more likely to take place if, from any cause, the ultimate tissues become inflamed, as in the various forms of catarrhal pneumonia or broncho-pneumonia. The lung loses its elasticity, its tissues are more open to infection, the residual air becomes stagnant, and its impurities, including foreign germs, are liable to be imprisoned for an indefinite time.

But that there is no necessary connection between ordinary diseases of the respiratory organs and phthisis may be shown by comparing the rates of mortality of these two classes of disease. I have here a chart (*see* p. 39), drawn from Lowe's Tables, from male populations at identically the same ages (*i.e.* from fifteen to fifty-five), from which it may be seen that the height of the line, indicating the prevalence of ordinary lung-diseases, bears no direct relation to the prevalence of consumption. It is true that both Liverpool and Manchester are

severely visited by both types of disease, but on each side of them stand many places in which there is no such correspondence to be seen.

MODES OF INFECTION BY THE TUBERCLE-BACILLUS

From what has already been said, it will have become abundantly evident that although we have not prejudged the question of contagion, yet that we regard tuberculosis as an infective disease, mainly deriving its origin from without the body, and that, in searching for its mode of entrance into the human frame, we must examine all the possible portals through which it can pass.

Now, there are four possible modes of access to the interior of the body: the skin, the generative organs, the digestive tract, and the lungs.

The first two are, I believe, comparatively rare sources of infection.

Tuberculosis is, indeed, inoculable by the skin, and there are on record cases in which it has thus been accidentally contracted. For instance, in Verneuil's 'Etudes sur la Tuberculose' (Paris, 1887), there are several such cases given, but the local disease did not spread to internal organs.

Cases of such generalisation of the virus are, indeed, occasionally heard of; thus, in the prospectus of the Congress held in Paris in 1888, reference is made to an experiment of Villemin's in 1869, in which a cutaneous surface, denuded by means of a blister, and powdered over with dried and pulverised tuberculous sputum, gave rise to general tuberculosis, and, again, in the discussion held at the Academy of Medicine in Paris (December 3, 1889) on prophylaxis against tubercle, Monsieur Nocard cited the case of a certain veterinary surgeon, named

Moses, who was wounded in the hand during the examination of the body of a tuberculous cow, and who at first had cutaneous tubercles on the cicatrix, and afterwards developed symptoms of phthisis, of which disease he died two years later.

These exceptional cases, however, only serve to show the rarity of the occurrence.

The most common mode of origin of scrofulous lymphatic glands is well known to be some previous lesion of the skin or mucous membrane, through which the virus has made its entrance; but, usually, the disease is arrested by the suppuration of the gland. Lupus, also, which is now generally acknowledged to be a tuberculosis of the skin, does not usually spread to the general system. On the whole, then, I believe that infection of general tuberculosis by the skin is a comparatively rare event.

Infection by means of marital intercourse is also probably very uncommon. It is impossible to say that it may not occasionally take place, especially in view of the observations of the French pathologists, which have already been quoted, as to the presence of the specific bacillus in the generative organs and in the semen.

FOOD

In former times the discussion of the question of the production of tuberculosis by food was limited to the subject of the adequacy of certain foods, and the quantity required to sustain the bodily vigour to such an extent as to prevent the tubercular diathesis, as it was called. But so soon as tuberculosis was recognised as an infective disease, experiments were made that proved the possibility of the entrance of the disease through the digestive

tract. Both Villemin and Cornil succeeded in producing tuberculosis by feeding animals with tuberculous material, and their researches have been confirmed by others, notably by Von Wesener. The question as to the suitability for food of the flesh of tuberculous animals is undoubtedly one of enormous importance. It has been stated (Dr. Behrend) that as much as 80 per cent. of the meat sent to the London markets came from such animals,[1] and in many places the prevalence of the disease amongst cattle has been found to be so great that it has been proposed, and in some places it has actually been decided, to place an absolute veto upon its sale.

Extreme differences of opinion, however, prevail as to the necessity for so greatly restricting the supply of animal food.

On the one hand, at the Congress on Tuberculosis, held in Paris in July 1888, M. Nocard urged that if an animal presented signs of tubercle in any organ, howsoever localised, it should at once be rejected as unfit for food, and he was followed by other speakers on the same side, with the result that a resolution was passed by the Congress 'that all meat derived from tubercular animals, whatever the gravity of the specific lesions found in them, should be seized and totally destroyed.'

Dr. Behrend, in a paper in the 'Nineteenth Century' review for September 1889, strongly supports this view, and quotes Dr. Klein in its favour, and ascribes the supposed immunity from tubercle enjoyed by the Jews to their careful selection of animals for food.[2] This view

[1] This must be an exaggeration, as the Departmental Committee on Tuberculosis in 1888 reported to the Privy Council that the proportion of tubercular disease among cattle slaughtered varies from 0·2 per cent. at Edinburgh to 50 per cent. (*Quest.* 4263).

[2] We have already seen that such immunity is by no means general.

also is not without some experimental evidence on its side, though Dr. Behrend does not mention it, for in Wesener's experiments swine and rabbits became, several of them, affected with tubercle, after being fed with the raw flesh of tuberculous cattle. Cooking, however, destroyed the virus, and dogs were unaffected.

Toussaint also succeeded in producing the disease by inoculating the juice from the flesh of a tuberculous pig. On the other hand Kastner,[1] after adopting most rigorous antiseptic precautions, injected the juice from the flesh of 12 beasts in all stages of tuberculosis, into the peritoneal cavities of 16 guinea-pigs, with absolutely negative results. My friend, Dr. Dreschfeld of Manchester, has made similar experiments with juice from the flesh of six cows, five of them in an early stage of tubercular disease, and one in an advanced form, though no tubercles were found in the flesh. The results from the first-mentioned group were entirely negative, the rabbits injected remaining perfectly healthy; but the juice from the flesh of the cow in a more advanced stage of the disease produced a small crop of tubercles at the place of puncture into the abdominal cavity of two rabbits after the lapse of from three to five weeks, and material from this tubercular growth injected into another rabbit again gave rise to still more decided tubercle.[2]

The evidence as to the transmissibility of tubercle by the ingestion of milk from tuberculous animals is much more distinct and positive as to its occurrence, even when there was no general infection in these beasts;

[1] *Münchener med. Wochenschrift*, August 20 and 27, 1889.

[2] Since this was written, these observations have been extended to ten cows in all; of these, six have now given negative results, three, in which the flesh was in an obviously unhealthy condition, have produced tuberculosis, and one, the flesh of which was apparently healthy, caused the disease after a lapse of three months.

and when we consider the enormous amount of mesenteric and visceral disease that exists at the present day amongst young children, it becomes a matter of extreme importance to the State that the bearing of this evidence should be made widely known, and that it should be followed by measures directed to avert the evil.

In the Registrar-General's Report for 1888, the deaths of 3,659 males and 3,115 females are ascribed to tabes mesenterica, and 3,722 males and 3,021 females to tubercular meningitis. Hirschberger declares that 25 per cent. of all children dying under one year of age die of tuberculosis.

It is assumed that many of these cases are directly the result of intestinal infection by the bacillus.

Bacilli have been found by Bang[1] in milk, both when the udders of the tuberculous cows were affected, and when they were not, and he obtained constantly positive results by feeding with the former, and occasionally with the latter, and he found that heating the milk to 70° C. did not always destroy its virulence. Only boiling would entirely do so.

Ferdinand May and Gottlieb Stein have made similar observations; but these researches are open to objection, on the ground that tuberculous material may have entered the milk from without; and, on the other hand, the digestive juices may act destructively on the infection when it is actually present.

On these grounds Dr. Hirschberger, under the guidance of Professor Bollinger, instituted a series of experiments in which extreme care was taken to avoid these possible sources of error. The milk was obtained in two ways: 1, by milking into glass vessels, sterilised by heat; 2, by drawing it directly from the udders of

[1] *Tidskr. f. Landoeconomi*, 1886.

recently slaughtered beasts, antiseptic precautions being strictly observed. Guinea-pigs were the experimental animals employed, and the milk was injected into the free peritoneal cavity. The animals were always young, and fresh on the market, and they were kept under excellent hygienic conditions. A very large number of the animals contracted tubercle, even when, as in these cases, the udder itself was unaffected, and although, out of twenty udders used, in only one was the presence of bacilli demonstrated by the microscope.

In only six cases was no infection obtained, and even when the cows were in otherwise good condition, which seven of them were, positive results were obtained in three cases.

He concludes from his researches that there is great danger attending the consumption of milk from tuberculous animals, in whatever condition they may be, and whether or not the disease is localised only.

Other observers, such as Von Wesener, Fischer, Baumgarten, Bollinger, Johne, and Martin and Queyrat, have also investigated the subject, and although they differ somewhat as to the exact track of the disease, they agree on the whole in regarding as dangerous the use of the milk of cows who are tuberculous, even when they only have the 'pearl disease.' Fischer and Baumgarten have shown, moreover, that the tubercle-bacilli are not deprived of their power for evil by the action of gastric juice, and Dr. Behrend quotes MM. Strauss and Wurtz as coming to a similar conclusion.

In the presence of facts such as these, we cannot deny the possibility of the transmission of tubercle by means of the flesh of cattle, and it is highly probable that mesenteric and intestinal disease is not unfrequently produced, especially in children, by the use of infected

milk, but there are certain broad considerations that should, I think, prevent us from attributing much of the phthisis that prevails to either source of infection, and this in spite of Bollinger's observation that the lung may become affected even when the tuberculous matter is introduced by the intestinal canal or the peritoneum; thus we do not find that the classes who eat most meat are those who are most commonly attacked by phthisis. Amongst our poor phthisical patients, how many there are who do not taste butcher's meat from year's end to year's end, and again, when the lower orders are able to procure this luxury, it is well known that they prefer it over-cooked rather than underdone; and the persons who, as we know, are most subject to tape-worm, from picking at raw meat, namely butchers and cooks, are far less liable to phthisis than other classes of the community.

It would be interesting also to know how those who ascribe such dire results to the eating of the flesh of oxen would account for the fact that in India, the Hindoos, who abjure such meat, are as subject to phthisis as the other races;[1] and also why the Gauchos of South America, who subsist almost entirely upon beef, are so remarkably free from the disease. Dr. Thorburn, of Toronto, tells me that the North American Indians, who now get no meat, are terribly subject to consumption.

Somewhat similar remarks apply to the use of milk, for the poor denizens of our towns, amongst whom phthisis and mesenteric disease are most common, are often unable to procure milk; but it must be acknow-

[1] Lombard, *Climatologie Médicale*, iv. 417: 'L'on voit dans les hôpitaux de Calcutta des phthisiques européens, hindous, mahométans et juifs.' I have already pointed out the tendency of the Jews of Alexandria to contract the disease. See Virchow's *Archiv*, 1883 or 1884. Schottelius

ledged that when they do obtain it, it is more likely to be obtained from diseased animals, and also more open to contamination by tuberculous dust in the small milk-shops in which it is stored. Still the comparative rarity of the disease amongst the children of the rich, who consume it largely, and who very seldom will take it after it has been boiled, shows that the danger from this source is not quite so serious as it has lately been represented to be, both in this country and on the Continent.

The measures to be be taken for averting the danger will be indicated in the next lecture.

THE ATMOSPHERE

The next medium through which the bacilli may be conveyed is the air.

We have already seen the close connection that exists between foul air and tubercular lung-disease, and we have studied some of the circumstances in which the bacilli may find a resting-place in the lungs, and under which they may develop into true tuberculosis.

But we have not yet determined the exact nature of the media through which these organisms are conveyed into the lungs—1, whether it comes directly from mouth to mouth, or 2, whether it comes indirectly from the sputum of phthisical persons—dried up, for the most part, and flying about in the air of dirty rooms or crowded assemblies in the form of dust. And the further questions arise whether the bacillus is kept alive, or its virulence increased, outside the body, either by the presence of aqueous vapour, or by emanations from the soil, or by other organic impurities connected with certain localities or certain dwellings.

1. *Direct contagion*—from mouth to mouth. In dis-

cussing the communicability of phthisis there has been a want of precision in putting the question as to whether direct or indirect contagion has in certain cases caused the disease. There can be little doubt, however, that, in the minds of most of those to whom the question has been put, it was the first or direct form in which it has presented itself.

The word contagion implies transmission by contact, and in the case of phthisis it would naturally be taken to mean the direct transference of the disease from person to person—in the words of Timon of Athens that 'Breath infects breath.' [1]

That contagion from a consumptive patient is a possible event, is an opinion that has been held by many eminent men—Morgagni, Van Swieten, Valsalva, Morton, and Baumé amongst the ancients; Laennec, Copland, Bowditch, Dr. Wm. Budd, Dr. Gueneau de Mussy, and Dr. Hermann Weber in more modern times. All these observers have expressed their view that the disease is contagious; and although there are many others on the opposite side, affirmative evidence is, in this case, of more value than negative. In Italy and Spain, as is well known, the disease is treated as contagious, and after death the bedding is destroyed and the dwelling thoroughly fumigated and cleansed; quite recently, too, a number of supposed cases of contagion have been contributed by medical men to the Collective Investigation Committee of the British Medical Association; and on the first perusal of the 'Collective Investigation Record,' it would seem to be impossible to doubt that, although the contingency may be a rare one, the possibility of such direct infection has been proved. I have myself shown, however, that the im-

[1] Act iv. sc. 1.

portance of these cases is liable to be exaggerated, and that they afford but little certain proof of the direct contagiousness of phthisis. In a paper on 'The Limits of the Infectiveness of Tubercle,' I have pointed out that—when checked by means of Dr. Longstaff's formula, supplied by the Committee themselves—the number of cases of phthisis in husband and wife, supposed to be due to contagion, is less than it would have been, had only accidental and non-contagious cases been admitted into the record.

Moreover, we have to take into account the possibility of contagion by sexual intercourse;[1] and the certainty that, in most of the cases of supposed direct infection, all the persons whose cases are noted were exposed to the same risks arising from unhealthy environment; and again, if phthisis were directly contagious, it is remarkable that it should be chiefly contagious in badly ventilated houses, and on certain soils; and it is further difficult to see why drainage of the land, and elevation above the sea-level, should in any way affect its contagiousness. Even good ventilation could hardly prevent a husband and wife, or other bedfellows, from communicating the disease, and yet such an occurrence has rarely, if ever, taken place in well-ventilated houses. In the Report of the Collective Investigation Committee, Dr. Dewar of Arbroath makes the significant remark, that in all his cases of supposed infection (No. 166), 'the patients lived in small, confined houses, and slept in the "box-beds" in use in Scotland.'

[1] It may perhaps be regarded as favourable to this view, that in 188 cases reported to the Collective Investigation Committee, 119 are of supposed transmission from husband to wife, and only 69 from wife to husband (*Report*, p. 33), and Dr. Weber's experience is to the same effect.

'During twenty-five years,' he says, 'I have not seen one case of contagion in the airy houses of the well-to-do.'

It is worthy of note that most of the physicians who have had the widest experience of phthisis are non-contagionists; thus Dr. C. T. Williams pronounces strongly against it, and gives statistics from the Brompton Hospital which are entirely opposed to the theory of contagion.

Dr. Pollock also says,[1] 'It can be shown that after an experience of thirty-six years, during which the hospital has been established, not only has no infecting process been evidenced, but that the medical officers and nursing staff, and the officials and servants, have been unusually free from phthisis.'

Dr. Andrews does the same from the City of London Hospital for Diseases of the Chest, and in his Lumleian Lectures for 1884 adduces several of the considerations against the theory, which had already occurred to myself before seeing his lectures. He concludes that 'there is not sufficient evidence to prove that the prevalence of phthisis is especially affected by direct contagion.'

Dr. Bennet, after twenty-five years' experience, says,[2] 'that if there has been any spread of phthisis at all by means of contagion, it has occurred very seldom, and only in quite exceptional circumstances.' In his summing up of the evidence on this point, Hirsch remarks[3] 'that contagious transmission of phthisis plays but a subordinate part in the spread of the malady.'

Baumgarten says,[4] 'Der Ursprung durch Inhalation, als Regel in hohem Grade unwahrscheinlich erachtet

[1] Pollock, *Lancet*, April 1883.
[2] *British Med. Journal*, 1884, ii. 704.
[3] *Handbook*, p. 232.
[4] *Lehrbuch der path. Mykologie*, p. 622.

werden müsste.' Ricochon[1] answers the question, 'Can contagion arise directly by respired air?' by saying that 'Experience is not favourable to this idea.' Verchère[2] holds the same opinion, and many other quotations of a similar import could be brought forward.[3]

2. As to the dissemination of phthisis by means of dust, it is well known that Koch regards this as the most common way in which it is produced—by the drying up and pulverisation of matter expectorated upon the ground, or on the floors of dwellings, or on handkerchiefs.

Cornet[4] has also warmly taken up this view, and by a series of experiments upon animals has proved the infectious qualities of dust obtained from the walls in the immediate neighbourhood of phthisical patients. In one case this dust produced tuberculosis, when removed from the wall of a room, six weeks after the death of the consumptive woman who had lived in it. In another, a room at an hotel was proved to be thus infected, and he points out the danger of infection from matter expectorated on the ground, or into handkerchiefs, and even from the dust of books in lending libraries.

Long before the discovery of the bacillus of tubercle

[1] *Etudes sur la Tuberculose*, Verneuil, 1887, i. p. 616.

[2] *Des portes d'entrée de la Tuberculose*, Thèse, Paris, 1884.

[3] Francis Galton (*Natural Inheritance*, p. 180) points out that, amongst the families from which his statistics were derived, only eight of the consumptive fathers had tuberculous children, and sixteen of the mothers; and he remarks: 'So far as it is contagious or infectious we must recollect that the child is peculiarly exposed during all the time of its existence before birth to contagion from its mother. During infancy it lies in her arms and afterwards lives much by her side, closely caressed, and breathing the tainted air of her sheltered rooms.' The explanation of the fact that we have been discussing appears, therefore, to be summed up in the single word 'infection.' But this does not exclude indirect infection from dust.

[4] *Zeitschrift für Hygiene*, Band v. 1888, 191.

also, this view was taken by some of the most competent observers: thus, Dr. Farr, in his Report to the Army Commission, remarked that 'the prevalence of phthisis in the armies of Europe is probably due, in part, to the inhalation of expectorated tubercular matter, dried, broken up into dust, and floating in the air of close barracks.'

Mr. Welch, in his prize essay 'On the Nature and Varieties of Destructive Lung Disease amongst Soldiers,' says, 'The chief deleterious agent in the generation of consumption is the organic matter, which, taken into the air-passages, there lodges and chronically irritates.'

Dr. Parkes apparently held the same view, for he speaks of 'phthisis and other lung-affections, which may reasonably be believed to have their origin in the constant breathing of air vitiated by the organic vapours and particles arising from the person.'[1]

I have ventured to ascribe most of the cases mentioned in my paper on 'Tubercular Infective Areas,' and especially the double and treble occurrence of the disease in the same houses, to this source of infection.

The retention of the bacillus either in dust, or in the vapour of the air, would fully account for most of the cases of supposed direct infection recorded by the Collective Investigation Committee, and especially for those in which the virus appeared to be introduced from without into houses previously entirely free from the disease.[2] It accounts fully for all the additional cases mentioned by Dr. Burney Yeo, in his able defence of the doctrine of contagion in the 'British Medical Journal'[2] (April 18, 1885).

The following case recorded by Dr. Niven would also

[1] *Practical Hygiene*, 6th ed., p. 135.

[2] See Cases Nos. 188, 194, 196, and 255.

come into the same category.[1] 'A married woman from Oldham, went with two children (one a lad æt. 10) to Southport, to join her husband, in September, 1887. The husband, wife, and the two children all slept in one front room. In the same house was a woman suffering from consumption, which was said to have been contracted from her husband, who had previously died of the same disease.[2] As the weather got colder, this consumptive woman asked them to change rooms with her, as her bedroom was colder than theirs. This was done. The Oldham woman used to complain of a peculiar odour in the room; it does not seem to have had any special cleansing or disinfection. In January 1888, the lad, æt. 10, died of "inflammation of the brain;" he had been ill a month. From the history, his fatal malady was almost certainly tubercular meningitis. The Oldham woman became *enceinte* in Southport, but removed shortly afterwards to Oldham, and was there confined in June 1888, but before the birth of the baby, she lost flesh, and showed the first symptoms of phthisis. The baby died in June 1889, of tabes mesenterica, and she died in October of phthisis.'

The terrible mortality from phthisis that has been observed in convents is most probably to be accounted for by the combination of bad ventilation, and want of perfect cleanliness, and of care in the removal of sputum-laden dust.

Laennec himself, in his classical work on 'phthisis pulmonalis' gives an instance of this in a religious community of women kept in strict seclusion and with sombre surroundings, amongst whom he had seen consumption arise so frequently, that in ten years' time the population of the institution had been two or three times renewed,

[1] *Public Health*, ii. 206. [2] This woman is since dead.

owing to the mortality of the inmates, with the significant exception of those who had charge of the gardens, the kitchens, and the infirmary.[1]

But the most striking series of statistics on this point is given by Dr. Cornet[2] who, by an elaborate statistical inquiry into the mortality from phthisis amongst the Catholic Nursing Orders in Germany, shows that the deaths from tuberculosis form nearly two-thirds of the total deaths, and in half the returns this proportion was exceeded, and in some mounts up to three-fourths. In two small mother-houses every death was put down to tuberculosis. Dr. Cornet ascribes this fearful mortality mainly to infection from dried-up sputum.

3. We have now reached, perhaps, the most difficult part of our inquiry—as to whether any conditions outside the mere presence of the bacillus are necessary, or at any rate auxiliary, to its infective power.

We still need an explanation of the facts: (*a*) that free ventilation is antagonistic to its influence, and that,

[1] The following case is given by Dr. Beddoes in his *Hygeia*, upon the authority of Dr. Luzuriaga:—

'A nun died at Bilboa of consumption: the furniture was burned, the walls, ceiling, and door washed, the floor taken up and sand laid in its place. The cell was then tenanted by another nun, in perfect health and of an excellent constitution. In two months she began to decline, her flesh wasted, the chest was attacked, the cough became worse and worse—in short, she died consumptive in eight months after taking possession of the cell. General purification as before. A third healthy nun succeeded and died in the course of a year. New examinations took place, and it appeared that the cord near the bed, attached to the dropping-bolt of the door, had not been removed. This, it was said, was impregnated with the sweat of the patients and with other noxious exhalations. It was removed, new furniture introduced, and a fourth nun has lived in the same cell for five years.'—Essay VII. p. 95.

The 'London Medical Record' for July 1884 quotes a similar case by Dr. Kempf, of Louisville.

[2] *Zeitschrift für Hygiene*, Band vi.

under this condition, it does not spread from person to person under circumstances of the closest possible personal contact.

(*b*) That its prevalence is greatly affected by a certain elevation of the site of dwellings.

(*c*) That it is more deadly and rapid in its action in hot, damp climates, and less common in cold and dry countries.

(*d*) That dampness of the subsoil under dwellings has a distinctly favourable influence in promoting its attacks.

(*a*) With regard to the influence of free ventilation, it is not only possible, but highly probable, that in many of the places where its good effects had been observed, other conditions as well have come into operation, and especially greater cleanliness and more complete removal of dust and other refuse. Where people care for fresh air, they also frequently show a preference for cleanly surroundings, both of house and clothes and person.

It is true that this consideration is not universally applicable, there are many very dirty dwellings that are well ventilated, as in the wattled huts of the Labrador fishermen, or the loosely constructed dwellings of the island of St. Kilda, and yet the inhabitants are almost exempt from the disease; but in these cases, apart from any disinfecting action of the air, or peat smoke, it is open to us to suppose that a good deal of the infective dust is swept away by the currents of air.

It may thus be possible to account for the absence of indirect infection through the medium of dust, in these places; though, as I have before remarked, it will not explain the non-occurrence of direct mouth-to-mouth infection, which may therefore be considered as rarely, if ever, operative.

(*b*) Somewhat similar remarks may apply to the in-

fluence of an elevated site upon even indirect infection through dust, for in these situations we probably have both more movement of air and less dust. We may also suppose that the health-giving properties of such sites would give to the dwellers upon them more power to withstand the bacillus, so that they could take in with impunity larger doses of these infective organisms.

In most cases, too, the rocks, or the subsoil, on which the houses stand, would supply to their inmates a purer and less vaporous ground-air than in less elevated regions.

(*c*) But no such explanations as these will touch the remaining two points that we have to consider. If there is no condition outside the body that influences the viability or virulence of the bacillus, it is very difficult to account for the comparative immunity of frosty or sub-arctic regions, and for the intensity of the tubercular action in hot or tropical climates. It is certain that chronic inflammations of the lungs are not more common in the latter, and thus one source of the disease is not so frequently present, whilst in the cold regions, where these affections are rife, phthisis is comparatively rare, and much less virulent.

We have no reason to suppose that bacillus-laden dust is absent in the crowded towns and close rooms which are to be found in Canada, for instance, and yet the disease does not spread as it does in the warmer and more genial air of Italy and the tropics.

(*d*) Again, it affords no explanation of the influence upon such infection of damp or undrained subsoils. Nor yet of the improvement in the phthisis-rate that has been shown to follow thorough drainage of a town.

As Dr. Andrew points out,[1] 'It is difficult to under-

[1] Lumleian Lectures, *British Medical Journal*, 1884, i. 659.

stand in what way the drying of the soil . . . could have so profoundly and so rapidly altered the constitution, habits, and vitality of the people of Salisbury, for instance, and of other towns, as at once to reduce, by a very large percentage, the number of cases of a developmental, or directly contagious, disease.'

To account for these several points, it is necessary to assume the existence of some conditions accessory to infection by the bacillus, which shall either enable it to live longer outside the body, or which may even increase its power of attack.

It may, indeed, be sufficient to account for the facts, if we assume the first of these hypotheses to be true, and if it could be proved that a hot climate, and a damp soil, give a longer term of life to the bacilli contained in foul and dusty air, for this circumstance would increase the probability that some of these organisms would find a suitable host; but I am inclined to think that something more than this is necessary, to account for the difference in intensity of bacillary infection in pure air, and that reeking with organic vapours.

I have, therefore, ventured to suggest that the tubercle-bacillus, like some other infective microzymes, may actually increase in virulence during a sojourn for a time in some medium external to the body—either in polluted ground air, or in an atmosphere saturated with aqueous vapour from the lungs.

There is nothing in the natural history of such organisms that need run counter to this theory, and as it has been shown by Dr. Thiersch and Professor Burdon-Sanderson, that the intensity of the poisons of cholera and enteric fever increases after their extrusion from the body, so it may be possible that contact with a certain kind of organic matter may assist the sporulation of the

bacillus of tubercle, and render it more infective after a time, than at the moment of its exit from the lungs of a phthisical patient.

I do not know whether the same observation has been made by other physicians to consumptive hospitals, but it has certainly struck me that a fresh infection of the lung, in persons already suffering from phthisis, is much more likely to take place from without, than from within the body. Many times have I seen in-patients with sputum teeming with bacilli, improve in health, gain weight, and almost lose the physical signs of their disease, and then they have returned home and in a few weeks they have come back to the hospital, sometimes with fresh disease in the damaged lung, sometimes in the opposite lung that had been previously healthy, and sometimes with laryngeal phthisis.

Such facts as these certainly seem to show that there was something in the air of their own homes much more infective than the bacilli of which they were themselves the hosts. The one fact that might seem to bear against this theory is the high temperature required for the development of the organism. And the greater virulence of the disease in hot climates, probably shows the influence of this condition, but it is by no means proved that, in cold climates, the bacilli contained in damp and foul dwellings may not in some way increase their power for evil.[1]

[1] On this point Dr. Candler remarks: 'I demur to the somewhat authoritatively pronounced dictum that it follows that the bacillus is a pure parasite from the one fact that it could not be got to grow outside the organism, except at blood-heat. The inference is by no means an inevitable sequence from the fact, for the fact does not take in enough to found such a weighty conclusion upon.

'It simply amounts to this—that the development-process of the bacillus did not occur at ordinary temperatures during the highly artifi-

I will conclude with yet one more striking case, quoted also by Dr. Andrew, from Mr. Fleming,[1] which it seems to me impossible to explain without some such hypothesis.

'A large farmer at Leinheim (in Alsace) for five years in succession lost one cow from phthisis, and always in the same stall. A sixth animal, selected for its vigorous health, and the absence of any taint of phthisis in its pedigree, shared the same fate. Then all the woodwork of the stall was removed, the manger and rack thoroughly disinfected, and the spot left unoccupied for some time. It was then re-built and re-occupied by several animals in succession, but tuberculosis made no more victims amongst them or in any other part of the stable.'

cial cultivations required in Koch's investigation. And one has to suppose, from the inference drawn from it, that this fact was regarded as equivalent to proof that the bacillus cannot be cultivated at ordinary temperatures under any conceivable artificial conditions. It would seem to have been assumed that because it did not grow in one series of experiments it was a certainty that it would not grow in any other series. But, as all possible combinations of soils and conditions were not exhausted in Koch's research, who shall say that some other investigator, or Koch himself, may not yet successfully cultivate this bacillus at ordinary temperatures.

'Again, even if it be granted that it shall be found impossible within the next ten or twenty years to cultivate the bacillus below blood-heat, it would not follow of necessity that there might not be an undiscovered means of growing it, as anyone who knows the history of the common yeast-form will readily apprehend. Moreover, if this bacillus should defy the efforts of man during all time to cultivate it below blood-heat he may nevertheless have been contributing, undesignedly, to its growth, and it may have been growing luxuriantly all around him, in the flora of temperate countries.

'Therefore I submit that Koch's premiss in this matter does not sustain the conclusion, and that the whole question of the botanical position of the tubercle-bacillus is an open one.'—*The Prevention of Consumption*, p. 10: Kegan Paul & Co.

[1] *Med.-Chir. Review*, October 1874.

As Dr. Andrews remarks, 'in this case, the disease must have been transmitted by some non-volatile substance in the stall itself; otherwise, and if the breath of the tainted animals had been infectious, it is difficult to believe that its neighbours on each side, for six years, could all have escaped unhurt.' . . . . 'It may have been an instance of local, not of personal, infection.'

LECTURE IV

THE PREVENTION OF PHTHISIS

Preventible causes: (*a*) inherited; (*b*) acquired; (*c*) sources of infection; (*d*) favouring conditions—Deterrent measures against marriage—Treatment of children of phthisical parents—Hygienic—Educational—Medicinal—Choice of work—Ventilation and warming of schools—Selection of subjects of acquired predisposition from chest diseases—Prevention of dusts from work—Change of climate—Legislative enactments respecting tubercular cattle—Flesh meat and milk—Combination of sanitary authorities—Measures respecting buildings—Compulsory clauses—Measures against atmospheric impurities—Healthy homes societies—Treatment of excreta—Sputum—Disinfectants—Combustion—Measures after death—Notification of phthisis—Isolation or supervision—Special hospitals—Principles of treatment of incipient phthisis.

We have now completed our survey of most of the conditions that are favourable or unfavourable to the development within the body of the bacillus of tubercle, and we have found that they may be briefly summed up under the following heads:—

1. *Congenital* taint or *inherited* proclivity to the disease.

2. Tendency to the disease *acquired* by unhealthy modes of life, and especially by such as produce actual lesions of the parts into which the organism can make its way.

3. Sources of possible infection, or means of access through which it may gain an entrance into the system and

4. Conditions that foster and keep alive the organism in the immediate neighbourhood of persons susceptible to the infection.

We are now in a position to consider what is possible to be done, and what should be attempted, to prevent the disease from attacking both human beings and animals associated in some relation or another with human beings.

1. With regard to inherited tuberculosis, or inherited vulnerability by the virus, if we were to accept Professor Baumgarten's theory of the chief mode of origin of the disease, namely by spores, that like the pangenetic 'gemmules' of Darwin, are implanted in us by our ancestors, and that may or may not remain latent all our lives, we should indeed despair of ever casting out this plague from our midst; for, even supposing that it was possible either to prevent all consumptives from marrying, or to keep their offspring under such conditions that the disease could not develop, it might still be transmitted through the second or third generation, and might break out at some subsequent period.

But it is quite otherwise with regard to inherited predisposition, if this consists, as we believe it does, mainly in a vulnerability only, and if, in nearly all cases, the actual germ of the disease comes from without the body. In this case it is quite possible to save even susceptible persons, by keeping them out of the way of infection, by clearing away the sources of the virus, or by doing away with the conditions under which it can exist, or by which it may be kept alive and ready to make its way into the system.

Doubtless, in view of the certain fact of inherited bodily qualities, and of the possible transmission of the disease congenitally, it is highly important to do what lies

in our power to deter consumptive persons from marriage. But when we cast about for the means by which this may be accomplished, it soon becomes evident that they are very few and inadequate. It would of course be quite useless to attempt legislation, for restrictive laws on this subject could not be carried out in this country.

Certain writers have indeed laid down an elaborate series of prohibitory rules, forbidding marriages of consanguinity, and between the members of tuberculous families (Ancell, 'Tuberculosis'); but even they acknowledge the impossibility of enforcing such rules. 'Love laughs at locksmiths,' and would certainly not submit to be bound by the chain of a prohibitory measure, even if one could be passed.

Still no one can doubt the importance of endeavouring to secure a healthy public opinion on the point, and fortunately most intelligent people are already, for the most part, on our side. Darwin's researches on the danger of breeding in and in, and on the advantages of cross-fertilisation, together with the general experience of the breeders of stock, on the advantages of the selection of suitable and healthy strains, and the patent facts relating to family inheritance of phthisis—all these things have combined to make the heads of families cautious in sanctioning marriages with individuals suspected of having a tuberculous ancestry.

Medical men and ministers of religion may do a good deal to strengthen this feeling, and to discountenance marriages between relations, especially when the family has been tainted by tubercle; also, by pointing out the facts that have been ascertained as to hereditary tendency to disease, they may often succeed in preventing the marriage of tuberculous individuals.

But when, in spite of all such checks, these marriages

have taken place, I believe—notwithstanding Professor Baumgarten's opinion—that much may be done to save the offspring from the disease.

A father or a mother whose partner has died of phthisis, and who has been left with children to bring up, may certainly hope to save them from the fatal inheritance, if due care is taken in their rearing and in their choice of occupation, and this is especially the case if the children have not been suckled by a consumptive mother. When this condition has been complied with, as it certainly should be in every case, the ordinary hygienic rules for infancy and childhood will, for the most part, suffice to keep the children in health; a well-drained, high, and airy site for the dwelling, entire cleanliness of house, clothing, and person, fresh air and light, abundance of good food, due exercise and rest, care during illness, and so on. I would, however, especially emphasise certain details of the hygienic code—thus, that milk and easily digested fats should form an adequate part of the dietary, and that the milk, in every case where there is a tendency to tubercle, should be scrupulously boiled, and in some cases peptonised, or used as koumiss or junket.

Free ventilation is especially needed in apartments occupied by persons, whether children or adults, with a tendency to consumption.

Dr. MacCormac,[1] gives a number of striking instances of the value of hyper-ventilation in warding off consumption. He also gives a graphic picture of his own practice in this respect. He says, 'I would speak in especial of a chamber which I once entered, as I had often before entered it, early one winter morn. It was the sleeping closet of my son. His low trestle-bed stood

[1] *Consumption and the Air Re-breathed*, pp. 109–112.

betwixt the severally widely-open window and door, while the keen but exquisitely fresh sweet atmosphere from wind-swept hills careered through the apartment ceaselessly. The hue of exuberant health mantled over the boy's every feature while, bordering the margin of the coverlet, there extended a fringe of pure white snow, which the genius of the fragrant night had wafted in all harmlessly during the hours of my child's repose' (p. 7). Truly it needs an enthusiast to carry a principle so far as this, but I should be glad if something very little short of it were adopted in every night nursery in the kingdom—if it could be said of all of them that

> Through the half-opened casements now there blew
> A sweet fresh air, that of the flowers and sea
> Mingled together, smelt deliciously.[1]

Then, as to exercise, it should be such as to develop the chest as much as possible—not only by games, though this is the best method, but also by carefully graduated and adapted gymnastic exercises. It should also, as much as possible, be carried on in the open air, and with as little dust in the atmosphere as practicable.

Especial care must be taken during and after the illnesses to which childhood is liable. After measles and whooping-cough thorough examinations should be made to see that there is no lingering affection of the lungs, and that the chest is not left deformed in any way. The attendants should not rest satisfied with anything short of complete recovery, if this can by any means be attained, either by medicinal agents, or gymnastic exercise, or by change of climate; and equal care must be exercised after any of the exanthemata. Where enlarged lymphatic glands are left, they must be regarded

[1] *Earthly Paradise*, ii. 281.

as a possible source of danger, especially when they are the sequel to some external eruption or sore.

Where the parents are rich, care should be taken with regard to education—that it should not be over-pressed, and that it should be carried on in well-ventilated rooms, and if a boarding-school has to be selected, it should be one on a high and dry site, and not only the school-rooms but the dormitories should be inspected, to see that there is sufficient cubic space, and also that the warming and ventilation are properly carried out. Whenever possible, it would be well to choose a cubicle or bedroom where the window can be kept open at night, winter and summer.

Let both boys and girls have an abundance of open-air life, and play games such as cricket and tennis, that promote healthy exercise; let them be allowed to walk and run, or ride, as much as they can consistently with other duties. Let the boys hunt, shoot, or row; let them camp out, climb mountains, or spend their holidays in the country, or at the seaside, in healthy lodgings.

If it should be necessary to choose a business or profession, let it be one in which there will be but little office work and as much out-door employment as possible. In such cases as these the prospect of future gain must not be allowed to compete with the pursuit of health, and hence, one with less remuneration and future emolument must often be preferred. Let a boy choose to be a farmer, a breeder of cattle, at home or abroad in some suitable climate, or a land steward or agent, or a surveyor or civil engineer. In a lower grade, other occupations may have to be selected, such as that of an agricultural labourer with all its hardships, a sailor, or fisherman, a carter, butcher, cab-driver, railway guard, or even costermonger. I well remember one

member of the latter craft who was in the third stage of phthisis, who had been attending the Addenbroke Dispensary for nine years, and whose disease certainly did not advance during the time that he was under my observation there—a period of three years—and such cases could be abundantly multiplied.

Something more needs to be done for the children of the poor by various public bodies, especially in the direction of securing better methods for the warming and efficient ventilation of schools.

Sir Henry Roscoe, in a recent paper, 'On the Ventilation of Schools,'[1] deduces from the researches of Professors Carnelly and Haldane, the conclusion 'that our primary schools are, as respects healthy atmospheres, in a deplorable condition,' and he thinks that the secondary schools and private advertised schools are far worse than board schools. 'We must not rest,' he says, 'until all our future elementary schools, to say nothing of the private schools, are ventilated by mechanical means, and the health of our children, physical and mental, is brought above the very low level at which it now stands.'

2. Many of the remarks now made will apply, at any rate in principle, to the subjects of an acquired tendency to consumption. Those who have been sufferers from any weakening disorder, such as have been enumerated as a cause of this tendency, will have to be sedulously guarded against the entrance of the tubercular poison into their systems, and they must be rigorously debarred from all crowded assemblies.

With regard to affections of the lungs, however, I think we may use some discrimination in selecting cases that must be submitted to the restrictions it is needful to impose upon those most prone to phthisis.

[1] Danks and Co., London.

Thus it is necessary that, during attacks of acute bronchitis, pleurisy, broncho-pneumonia, and other inflammatory complaints, care should be taken to secure the complete resolution of the disease; but when this has been attained, and the full elasticity of the lungs has returned, it has not been within my experience, that persons who have suffered from these complaints are more liable to consumption than other people, though of course subsequent attacks may at last leave the lungs more vulnerable by the disease.

After all these complaints, and especially after pleurisy, there is undoubtedly danger, so long as there remains any contraction of the chest, and want of full expansive power. It is well known how frequently phthisis follows pleurisy, and although this may in some cases be due to the fact that the pleurisy itself is of tubercular origin, in others the tubercular infection is grafted upon the injured organ at a period too remote from the original disease for it to have been its immediate precursor.

I have been in the habit of testing the movements of the chest with the stethometer in most forms of pleurisy, and in all cases where these movements are impaired by the attack I have formed an unfavourable prognosis, an opinion that has only too often been confirmed by the result, though in several cases the supervening phthisis has been some years before it has made its appearance.

There seems to be less reason to dread the occurrence of phthisis in chronic bronchitis, asthma, and emphysema, but I cannot altogether share the opinion of Rokitansky as to the immunity enjoyed by sufferers from the latter complaints, as I have several times seen their association with phthisis.

I should imagine also that in the partial emphysema

that arises from atelectasis or impairment of certain portions only of the lung, a deposit of tubercle is but too likely to take place in the portions thus injured.

Still greater danger is also acknowledged to arise from accidental hæmorrhage into the lung-tissue. Although hæmoptysis is frequently only a sign of incipient tubercular disease, I am convinced that it is also sometimes simply its precursor, and the resulting phthisis has in my experience been more acute in its course. In this class of cases also the stethometer has been of no assistance in prognosis. I may remark here that, as an additional precaution against infection in this class of cases, and in all in whom there is a predisposition to phthisis, and when its subjects are obliged to attend crowded assemblies of any kind, I have been in the habit of recommending the temporary use of respirators charged with eucalyptol, so as to minimise the danger of inhaling tuberculous material.

I need not linger over the precautions that should be taken in the case of dusty work, especially such as involves the production of sharp, irritating particles; working men are now much more alive to the danger arising from it than they were formerly, when, as Dr. Hall, of Sheffield, tells us, the file-grinder looked upon early mortality from lung-disease as inevitable, and rather welcomed it, as affording a chance of more speedy promotion.

It is probably still hopeless to expect that work-people will use respirators to catch the dust on its way to the mouth, but fans are now almost universally employed to sweep it away from the worker.

It would, however, be a good thing if these people could be more frequently warned of the necessity of attending to the first beginnings of lung-trouble, as we

know that, in their first inception, many of these cases are not tuberculous.

In all cases of incipient chronic lung-disease, wherever possible, an attempt should be made to obtain plenty of fresh air and sunshine, whether by change of climate or otherwise. I venture to put this matter in these terms because I believe that, in the treatment of such cases, wherever a dry, pure soil and germ-pure air exist, that is the best health-resort in which the largest amount of outdoor occupation can be secured, together with the greatest amount of bodily comfort.

Hence, it is that Davos Platz and San Moritz can vie with the Riviera and Algiers; Canada, with California and Mexico; the highlands of Africa, with the Canaries and Madeira.

3. With regard to the ingestion of tuberculous food it is probable that too great laxity prevails in the inspection of butchers' meat, and that its sale should be subject to more stringent regulations than it now is in our large towns. In France, by a decree passed in July 1888, it is provided that 'whenever the tubercular process affects the lining membrane of the chest or abdomen, the entire carcase shall be condemned,' but in most other countries it is considered sufficient, when the disease is entirely local and not generalised, to cut out the diseased parts and suffer the meat to be sold, if otherwise sound and the beast well-nourished. Professor Koch, himself, thinks there is no danger in this practice, and this opinion is shared by Cornil and Dujardin Beaumetz in France, by Nosotti in Italy, and by Von Wesener in Germany.

But most of these opinions are based upon the idea that the meat in question should be well cooked, and it seems to me desirable that the Prussian practice should

be followed, of plainly labelling meat that is regarded as in the least degree suspicious by the inspector, who should not suffer it to be sold without a caution to the buyer.

Still more decided precautions should be taken with regard to *milk* from tuberculous cows. Its power of conveying tubercle is fully proved, and its sale should be prohibited. In the meantime, also, as before said, the possibility of infection from this source should cause the heads of families either to make very sure as to the untainted source of the milk supplied to them, or should induce them to insist upon its being boiled before use.

In towns, it is impossible to trace the milk to its source, and the latter injunction becomes, therefore, more imperative. Probably, many more cases of infection from this source would occur in towns if it were not for the fact, pointed out by Bollinger, that the mixing of the milk of many cows diminishes, by dilution, the risk of conveying the disease.

The possibility of tubercle arising from this source, lays a heavy burden of responsibility upon those who have the care of the public health, and this burden, with regard to milk, has now been laid upon the Local Government Board and its subordinate local authorities.

By Section 9 of the Contagious Diseases (Animals) Act of 1886 (amending the Act of 1878), the powers of the Privy Council respecting the inspection of cattle, and sanitary precautions relating to milk, are transferred to the Local Government Board, and local authorities are empowered to make regulations respecting these points. Unfortunately, tuberculosis is not included in the definition of 'disease' in the Act, and the Departmental Committee on Pleuro-pneumonia and Tuber-

culosis report, in 1888, that 'although in England and Ireland, under the provisions of the Nuisance Removal Act (as embodied in the Public Health Act, 1885), the Medical Officer of Health, or Inspector of Nuisances, may seize tuberculous animals, yet such seizure is rarely performed.'

The case with regard to milk is even more unsatisfactory. It is, doubtless, now the duty of Rural Sanitary authorities to supervise all dairies and cowsheds, and to prevent the export of possibly tainted milk; but, again, there is no direct provision against the supply of the milk of tuberculous cows; moreover, the local boards of places where milk is produced are not necessarily in union with the authorities of the places where it is consumed, and the members of the rural local boards are often unwilling to offend the farmers, and others by whom they are elected, and are not likely to take much care in the matter.

What is needed is greater solidarity amongst the various local authorities, so that the corporation of a town, for instance, might be in communication, through its health officers, with the inspectors in the rural districts from which the milk is supplied to the townspeople. They might thus ascertain directly the presence of disease, whether of man or beast, in the farms supplying the milk.

But even County Councils cannot altogether perform the necessary duty of carrying out this organisation, for the milk supply of a town may come from several different counties. I know, for instance, that Manchester is supplied not only from farms in Lancashire, but also from those in Cheshire, Derbyshire, Yorkshire, and even Staffordshire.

It is urgently necessary, therefore, that the laws re-

lating to the sale of the flesh of tuberculous cattle, and the milk supplied from them, should be amended without delay, and also that some concerted action should be taken by health authorities with reference to the sale of milk in towns.[1]

4. The next, and I believe the most important, media for propagating consumption are the houses people dwell in, and the rooms in which they congregate together.

Architects and builders of all classes of dwellings have been in the past, and are still, largely responsible for a very large proportion of preventible mortality. It is only right that we should look to them in the future to rectify the mischievous modes of construction—both of mansions, and cottages, and public buildings of all kinds—that have proved such a fertile source of disease, and especially of consumption. It is incumbent upon them both to rectify existing buildings, and to erect new ones upon plans that are more in accordance with modern sanitary requirements. They must see to the exclusion of noxious ground-air from houses, to the prevention of the harbourage of dust, to giving sufficiently copious streams of air without draughts, and to the extraction of foul air as soon as it is produced, and to the provision of abundance of light. They are not left without help in this regard. In Sir D. Galton's excellent work on 'Healthy Houses,' there are ample and detailed instructions as to how these several objects may be accomplished.

[1] I am informed that several towns have obtained private Acts, by which it is provided that their judicial officers of health may inspect any dairies, &c., beyond their boroughs from which milk is supplied to them; and these provisions are included amongst the 'model clauses' which may hereafter be the subject of general legislation. But it is not apparent from these clauses that any power is given to inspect the cows themselves, in order to judge whether they are tuberculous or not.

Local authorities also cannot plead ignorance in excuse for neglect. The Model Bye-laws of the Local Government Board, if fully carried out, would to a great extent do away with the merely structural causes of phthisis. The whole of these bye-laws are useful; but those relating to the width of streets (4–8), height of buildings (19), space around houses (53–55), concreting of cellars and foundations (9–10), damp-proof courses, (17) quality of materials and fireplaces (11), (58), drainage (60), are of especial value in relation to our subject.

Much greater care should be exercised in respect to the warming and ventilation of public buildings. The openings for the latter purpose are for the most part quite inadequate, and the means of extracting foul air are often futile. It is a question also whether the methods used for introducing warmed air do not generally devitalise it and make it unfit for healthy respiration.

The Model Bye-laws, moreover, only apply to new streets and buildings, and it is in the old parts of most large towns that the favourite breeding-places of phthisis are to be found. Recent reports by Dr. Russell in Glasgow, and by Dr. Thresh in Manchester, show how entirely unfit are many of these places for healthy habitation, and gives evidence of the effects of overcrowding.

Dr. Thresh found, in Ancoats, that 'the cubic space per head in the living-rooms and bed-rooms in Ancoats is miserably inadequate for sustaining health;' in one case, a man and his wife, with five children, lived in one room—8 ft. 6 in. high, 10 ft. 6 in. long, and 9 ft. 3 in. wide—647 cubic feet in all; and he gives several similar cases in his Report to the Sanitary Association of Manchester.

These abominations in the shape of dwellings ought to be absolutely done away with—razed to the ground if they are incapable of alteration. Back-to-back houses should be converted into double-houses with thorough ventilation. Closed streets, courts, and alleys should be opened up, and decent habitable houses built in their places. Breathing spaces and playgrounds should be left in their midst.

Local authorities have ample powers now for dealing with all these matters.

Numerous Acts of Parliament have been passed since the first Labouring Classes Lodging Houses Act of 1851, all of them having for their object the improvement of the dwellings of the working classes. There was one in 1866, and another in 1867, and in 1868 a very stringent measure was passed, commonly called Torrens's Act, but unfortunately it proved unworkable, owing to the absence of clauses giving sufficient compensation to owners of property.

Then, in 1875, came the most important effort in this direction, in Sir Richard Cross's Improvement Act, and it must be a source of no small satisfaction to the members of this College to know that this and other subsequent measures are mainly due to the representations made by them to the Legislature as to the need for such improvement.

Thorough as this Act was, however, it also was not found to work well in practice, and several large towns, notably Edinburgh and Glasgow and Manchester, preferred to obtain private Acts of their own, to using the rather cumbrous machinery provided for them in it. Accordingly amending Acts were passed in 1879, remedying defects both in Torrens's and Sir R. Cross's Acts, and in 1881 further efforts were made in this direction.

Finally in 1885, after a Royal Commission had sat upon the subject and had reported that local authorities really now possessed ample powers—which for the most part they did not use—for improving cottage dwellings, a most important clause was introduced into the housing of the Working Classes Act 1885, which made compulsory all that was only left permissive before in previous Acts.

This clause runs as follows:—

'Section 7. It *shall* be the duty of *every* local authority entrusted with the execution of laws relating to public health and local government, to put in force from time to time, *as occasion may arise*, the powers with which they are invested, so as to secure the *proper sanitary condition* of *all* premises within the area under the control of such authority.'

If the local authority does not carry out this law, then any complainant may apply directly for a mandamus to compel them to do so, without the necessity of appealing first to the Local Government Board at Whitehall.

This is indeed an important clause, and if it is interpreted according to the lights of modern sanitary research, it ought to transform our large towns from their present condition of hotbeds of disease to a collection of smiling abodes of health.

In the matter of atmospheric impurities, cleanliness, light, and ventilation must be our chief resources.

The cleanly housewife's righteous horror of dust has a more scientific basis than she dreams of; as we have seen, it may be the vehicle of disease and death, and is something more than merely 'matter in the wrong place.' But with the exception of giving her this incentive to an incessant fight against it, I doubt whether we could

teach her anything as to the best mode of dealing with it. I should be glad, however, to see all furniture and room-fittings so made as not to be the harbourers of dust, with their edges bevelled off or rounded; and it would also be a good thing if the contents of the dustpan were invariably burnt at once, instead of being stored up in a bin, or carried off to form a huge mound in some town's-yard, many of its lighter particles being carried away by the wind, to add to the other deadly ingredients of a polluted atmosphere.

It is interesting to note, as a result of Professor Carnelly's experiments,[1] that the influence of dirt as a fosterer of micro-organisms in the air has now been placed upon a scientific basis. His conclusions are shown in the following table:—

TABLE VIII.—*Effect of Cleanliness.*

		Space per person	Carbonic acid	Organic matter	Micro-organisms
One-roomed houses	Clean	295	7·99	2·34	18
	Dirty	200	9·87	3·23	41
	Dirtier	221	10·66	2·42	49
	Very dirty	220	10·01	2·69	93
Two-roomed houses	Very clean	273	12·20	1·93	10
	Clean	264	9·34	1·37	22
	Dirty	233	9·40	2·03	69
Unventilated Board-schools	Clean	167	19·68	3·25	91
	Average	166	14·17	2·90	125
	Dirtier	191	22·47	2·73	198
Ventilated schools	Very clean	194	12·50	2·26	3
	Clean	155	12·81	1·48	16
	Less clean	152	10·78	1·75	30

It will be seen that cleanliness produced a difference of 18 to 93 in the one-roomed houses, 10 to 69 in two-roomed houses, 91 to 198 in unventilated, and 3 to 30 in ventilated schools.

[1] Sir H. Roscoe's Paper, p. 12.

And yet want of cleanliness is not solely responsible for the presence of organised germs.

Sir Henry Roscoe draws the following conclusions from Professor Carnelly's paper to the Royal Society :—

(1) That, 'by mechanical ventilation, the micro-organisms (that is, the excess over and above those contained in the outside air) were nearly one-tenth, the organic matter one-seventh, and the carbonic acid nearly one-half, of what they were in the schools ventilated by ordinary methods, the average cubic spaces in the two schools being practically the same, or, if anything, slightly less in those ventilated mechanically. (2) Notwithstanding this very great improvement in the purity of the air, the temperature is even considerably higher than in the ordinarily ventilated schools; whereas, to produce such an improvement in purity by the ordinary method of opening windows, &c., would have reduced the temperature to a very uncomfortable and dangerous degree. (3) This improvement is also obtained with little or no perceptible draught, and where a draught is perceptible, it is a warm and not a cold one, as is the case with ventilation by an open window. (4) Mechanical ventilation not only very considerably reduces the number of micro-organisms during the time that it is in action, but has also a marked effect after the ventilation has been stopped and replaced by natural ventilation, for the micro-organisms in the air do not rise to the level of the ordinarily ventilated schools for a considerable time.'

It would be well if, in framing byelaws for all kinds of public buildings, and especially for schools, the suggestions made in Sir H. Roscoe's valuable paper could be followed.

Another table shows that the micro-organisms are derived from the rooms themselves.

TABLE IX.

Cubic space per person	Naturally ventilated				Mechanically ventilated			
	No. of cases	Carbonic acid	Organic matter	Total micro-organisms	No. of cases	Carbonic acid	Organic matter	Total micro-organisms
Cubic feet								
50—100	6	21·5	16·2	119	—	—	—	—
100—150	14	15·5	19·6	128	7	14·0	7·8	23
150—200	5	18·9	12·3	150	8	11·4	9·6	14
200—250	9	21·1	16·8	188	5	11·8	12·3	10
250—300	4	17·1	9·5	187	—	—	—	—
300 & upwards	4	15·1	11·8	12	6	13·0	3·7	2

In badly ventilated schools, micro-organisms *increase* up to a certain point with increase of wall and door space, whereas, in mechanically ventilated schools, where the air is quickly removed, the micro-organisms *decrease* with increase of cubic space.

Sir Henry Roscoe says, 'We have already seen that cleanliness (see Table, p. 119) exerts a most powerful influence on the number of micro-organisms. One would expect that the source of these organisms is the floor, especially the dirt lying about on it.

But that the loose dirt lying about on the surface of the floor is not of itself the chief source seems to follow from two facts. In the first place, one would expect that scrubbing the floor would have a marked effect on the micro-organisms in the air. To test this, two similar schools were taken; one was scrubbed once a week for five weeks, and the other left unscrubbed. The bacteria were regularly determined in each, with the following result:—

TABLE X.

	Scrubbed room	Unscrubbed room
Average of six days before scrubbing	112	93
Average of 1st day after scrubbing	31·5	70·5
,, ,, 2nd ,, ,, ,,	133	86
,, ,, 3rd ,, ,, ,,	147	112
,, ,, 4th ,, ,, ,,	134	46
,, ,, 5th ,, ,, ,,	108	99
General average after scrubbing	111	83

The total effect of scrubbing also was found to be *nil*. The scrubbing would remove the loose dirt, but not the dirt ingrained in the floor.

Another reason against the loose dirt being a main source of contamination is the fact that the infection of a school with micro-organisms takes place very gradually, new schools having much fewer than old ones; and similar results have been obtained by Miguel in new and old houses in Paris. Probably nothing short of total destruction would clear some of the older buildings from micro-organisms.

Even if we suppose that all the precautions so far mentioned could be strictly carried out, much still remains to be done before we can exorcise the demon of tuberculosis.

People must be educated to take advantage of the arrangements that we may hope will be made for their comfort and well-being. At present, many of the inhabitants of both town and country are quite capable of converting a palace into a pigsty, and the best-laid plans for ventilation and cleanliness may not only 'gang aft aglee,' but may be deliberately frustrated by the ignorance, carelessness, or active prejudice of the people most concerned to carry them out.

These people will have to be taught the importance

of the sanitary regulation of their households, and to this end they must know something of the laws of health, and they should be assisted to make proper use of the various means that are placed at their disposal.

It would be a most beneficial thing if sanitary associations, or healthy homes societies, similar to those in Manchester, could be established in every town in the kingdom.

I have ventured to suggest to the Sanitary Institute the establishment of such societies, as part of its work in the towns where its congresses are held.

5. Hitherto we have been mainly occupied with the external precautions to be taken against absorbing the tubercular virus. We must turn now to those relating to the sufferer from consumption himself.

First, the treatment of the excretions from tubercular patients. Both the urine and fæces have been found to contain bacilli, and should therefore be disinfected, or otherwise safely disposed of; but we have seen that the most fertile source of infection in phthisis is probably the sputum of such patients expectorated on to the ground, on to floors, or into handkerchiefs, allowed to dry up, and, in the form of dust, permitted to lodge on the walls or in nooks and corners of dwellings, or to float about in all kinds of public assembly-rooms, whether for religious, political, or social meetings. Now we can say at once that this should never be permitted.

Englishmen are rather inclined to sneer at the abundant provision made on the Continent for the reception of expectorated matter, but it might be well for us if we were more cleanly in that respect in this country.

Undoubtedly, if the contents of these receptacles are simply consigned to dustbins, and left to gain virulence, as it is at least possible they may, by contact with organic

filth, then the danger of infection from this material may be but little diminished; for when it dries up and portions of it are allowed to fly about in the atmosphere of the streets and lanes of a city, it may spread the disease, through the medium of the very air that we admit to our living rooms in the laudable desire for ventilation.

But all such receptacles should be filled preferably with some liquid disinfectant, or with sawdust that has previously been charged with some efficient disinfectant, and their contents, like those of the dustbins, should be scrupulously burnt, either on kitchen fires, or in a furnace provided for the purpose by town authorities.

It would be well, also, if all phthisical patients would carry with them and use small portable spittoons, that could be carried in the pocket. They can be made for a few pence, and can be lined with paper charged with disinfecting substances, and their contents could readily be consigned to the fire at convenient intervals.

Disinfectants.—Numerous experiments have been made to determine the best means of disinfecting tuberculous material.

In Verneuil's 'Etudes sur la Tuberculose' Dr. P. Villemin gives the results obtained with a prodigious number of different chemical substances. It would be useless to name the hundred and one of these bodies that have either given negative results, or that have only somewhat retarded the development of the bacillus. He found, however, that the following ingredients mixed with the cultivating medium completely sterilised it in the proportions which he employed—they are: hydrofluoric and silicic acids, fluosilicates of potassium, sodium, and iron, ammonia, naphthol (α and β), polysulphuret of potassium, tartar emetic, and sulphate of copper.[1]

[1] Amongst the class of substances that greatly retard the develop-

Dr. de Souza found that the following substances prevented all development. Mercuric ethyle, in the proportion of 1 in 35,000; benzoate of ethyle, 1 in 3,000; benzoate of methyle, 1 in 12,000; but menthol and iodoform had only a retarding influence.

Schill and Fischer ('Mitth. aus dem k. Gesundheitsamte') found corrosive sublimate most efficient; Sormani and Brugnatelli[1] brom-ethyl, palladium and mercuric chlorides, carbolic acid, kreasote, naphthol, and saturated solutions of camphor in alcohol, lactic acid, and turpentine.

Unquestionably tuberculous material cannot be left to take care of itself. The various natural disinfecting agents, with perhaps the sole exception of fresh air and light, seem to be wholly inadequate to deal with it.

M. Galtier[2] has succeeded in inoculating tubercle with the juice of muscle, from tuberculous animals, after it has been raised, for twenty minutes, to a temperature of 60° C., and for ten minutes to a temperature of 71°, temperatures which, he says, are not exceeded in the centre of a large piece of meat cooked on the gridiron.

Dessication, putrefaction (for ten to twenty days), freezing at temperatures from 3° to 8° below zero (C.), alternate freezing and thawing—none of these conditions destroy the infective power of tuberculous matter.

ment of the bacillus, he places arsenious, boric, picric, pyrogallic, and sulphurous acids; benzine, chloroform, creasote, hyposulphite of soda, iodoform, menthol, phenate of soda, salol, and toluene. And he places in a third category those substances in which growth took place, but with some difficulty; amongst these we find, acetone, aldehyde, biniodide of mercury, bromides of potassium, sodium, and ammonia; caffeine, camphor, chlorhydrate of ammonia, turpentine, eucalyptol, iodide of potassium, naphthaline, resorcin, sulphates of soda, magnesia, quinine, and zinc, thymol, and tungstate of soda.

[1] *Ann. Univ. di Med. et Chir.* xxvii.

[2] *Comptes Rendus*, July 1887, 105, p. 231.

He found also that the urine of tuberculous animals was virulent, and he draws the conclusion 'that it is indispensable to insist upon the disinfection of all objects soiled by tuberculous animals, of their excretions, of places they have occupied, of the dung-heaps, &c., in order to prevent the dissemination of the disease, and its transmission to man.'

At the Manchester Hospital for Consumption we have been in the habit of using for the spit-cups a solution of mercuric chloride, 1 in 500, and I have also used 'salufer,' a fluosilicate, introduced by Mr. W. Thomson, of Manchester.

If the stools and urine of phthisical patients have to be disinfected, probably the best agents would be sulphate of copper, or sulphate of iron, or carbolic acid; but where water-closets are used, there is probably not much danger from this source.

It is recommended by Dr. Cornet and others that after the death of a phthisical patient, the rooms, and bedding, and clothes should be thoroughly disinfected, as after a death from a contagious disease, and a thorough cleansing of the whole premises.

There can be no doubt that this practice—at present, so far as I know, limited to Italy and Spain—ought to be universal.

If all the suggestions that have so far been made were to be carried into effect to any large extent, we might, I think, fairly hope that in a few years there would be a considerable reduction of the phthisis death-rate; but, after all efforts in this direction have been made, there will probably still remain a large number of persons, actually suffering from the disease, who will escape from the net of precautions that we have endeavoured to cast around them; and in their heedlessness

and contempt of danger to themselves and others, they will continue to spread about the seeds of disease, and thus susceptible individuals will still continue to contract the complaint.

It becomes a serious question for the public and for the State, whether persons suffering from consumption, if they are 'without proper lodging and accommodation' for preventing infection, should not be provided with asylums or hospital accommodation, until the disease is either cured or ends in death.

Enormous sums have been spent in the past, in the isolation of persons suffering from leprosy, a disease strictly analogous to phthisis, only still more chronic, and less easy to control by such a method. It would certainly be far easier to cleanse away and destroy phthisical sputum, than to prevent contamination from leprous sores.

If a poor country like Norway can provide asylums for a large proportion of its lepers, it is not too much to ask Great Britain to make the attempt to segregate those consumptives who are likely to be a source of danger to the community.

At the present time, the accommodation for cases of phthisis is very small. They are entirely shut out from most general hospitals, although they are admitted as out-patients to all dispensaries, and are thus free to scatter abroad the infective material in places where many persons peculiarly prone to the disease are sure to congregate.

The special hospitals for consumption are very few in number. They may almost be counted upon one's fingers: thus there are in the metropolis four such institutions, including the great Brompton Hospital, and together containing 615 beds.

At Bournemouth also there is the auxiliary sanatorium with 62 beds.

At Torquay (48), Ventnor (140), and St. Leonards (18), there are special hospitals, with an aggregate of 206 beds.

Possibly, also, at some of the numerous cottage-hospitals, cases may occasionally be taken in.

The only manufacturing towns in England to which such hospitals are attached are Liverpool, with 50 beds, Manchester with 37.

The workhouse hospitals are the chief refuges for destitute consumptives; but Dr. Bridges informs me that there are no data from which to ascertain how many of them are thus accommodated.

In any case there are certainly large numbers of persons suffering from active disease who are without proper means of treatment, and who are permitted to mix freely with the rest of the community.

I am well aware that, at the first blush, these proposals will be regarded by most people as stupendous, and perhaps as entirely impracticable; and if it was intended to provide for even a moiety of the consumptives of the country, they might rightly be so regarded. It would, in this case, mean an expenditure of several millions; and even if this amount were forthcoming, the social difficulties in the way would be still greater, for it would involve the separation of families, and, moreover, many consumptives are the sole bread-winners or guardians of families, and could not leave them.

But I do not seek for anything so gigantic as this would imply. I would simply ask that phthisis should be placed in the same list with other diseases requiring special measures to prevent its spread. Although, as before said, phthisis is not directly contagious, I venture

to contend that there would be nothing unreasonable in thus classing the disease. Under the recently passed Notification of Diseases Act, many local authorities have included enteric fever in their schedules—a disease strictly analogous, in that it is rarely, if ever, directly infectious, but which spreads mainly by means of excretions from the patients.

There would also be nothing strained in interpreting the words of the Public Health Act, 1875, in the clauses relating to infectious diseases, 'without proper lodging and accommodation,' so that they should mean that special measures must be put in force to prevent people from sowing broadcast the virulent particles coughed up from their diseased respiratory organs.

My proposition briefly would be this, that phthisis should be classed with other infective disorders; that every case, as soon as it is discovered, should be notified to the Medical Officer of Health. If necessary, it should be visited, to ascertain whether proper care is, or can be, taken to prevent injury to the public health. Where the case is that of a poor person, the local authority should see to the regular cleansing and whitewashing of the premises, and to the disposal of excretions, including especially the expectorated material. They should also inquire into, and rectify if necessary, the drainage and ventilation of the dwelling; and after death, special measures should be taken for the cleansing and disinfection of house, bedding and clothes.

After all this had been done for the safety of the non-phthisical portions of the family, there would next come the question of the propriety or possibility of removing the sick person to hospital. So long as he (or she) could work, and so long as he would consent to use the necessary means for destroying the infective

material, it would be unnecessary to do more than I have already indicated; but when the patient becomes unable to follow his employment, and the family are obliged to seek for assistance from the parish, he has a claim to be received into the workhouse hospital, and such an asylum should be offered to him, and should be made as little humilitating and as free from ignominy as possible. When such a hospital is efficiently administered, as it should be, and often is now, when the nursing is performed in a kindly fashion, on Miss Nightingale's principles, and the patient is not left to the ministrations of any able-bodied pauper, as is sometimes the case; and when appropriate care is taken for the cleanly disposal of excreta of all kinds, then there could probably be no better fate in store for the poor invalid, and he might even soon be restored to his family, and be able to return to his work.

I would also put in a plea for those who are not reduced to pauperism, but who could be removed to hospital to receive appropriate treatment in its wards. Towards the close of their illness, persons who live in close, confined dwellings become a serious source of danger to the rest of the family, and as they are 'without proper lodging and accommodation' for the safe treatment of such a disease, I would submit that it would be a legitimate expenditure on the part of local authorities, if they were to provide male and female wards for the reception of such cases, in connection with their hospitals for infectious diseases. Although consumption is not directly infectious, its products are undoubtedly infective under certain conditions such as have been mentioned, and local boards would be taking the right measures for preventing the spread of disease if they were to make such provision. There are probably few

exanthematous diseases that could be so easily and effectively controlled.

But there would still remain a wide field open to private benevolence in the provision of sanatoriums or homes for these unfortunates.

I can conceive few objects better calculated to excite the compassion of all humane people than the condition of many of these poor sufferers, who are shut out from the benefits of general hospitals.

Most people have had an opportunity, some time in their lives, of watching the course of this insidious malady, and have witnessed the scenes of weakness and suffering that attend its later stages. And if it is painful to contemplate its ravages in those who are well-to-do, and surrounded by all the alleviations that can be brought to bear by medical science, how much more sad must it be in the homes of the poor, with scanty attendance, imperfect nursing, and with a lack of all kinds of comforts for the sick.

I can scarcely think that there would be much difficulty, in raising the funds necessary for the care of such cases as are not yet reduced to pauperism, but who are really destitute of the appliances and comforts so necessary to a slowly fading life.

The chief difficulty in the way would lie with two classes of objectors.

1. There are some practically-minded men who are not destitute of benevolent impulses, but who are wishful to see the useful application of the money they are willing to expend. They may have hesitated in the past, and may reasonably still hesitate, to give anything towards an object which they consider is tantamount to being merely an attempt to smooth the pillow of the dying. These men look upon hospitals for con-

sumptives as asylums for the dying, and they would write over them Dante's inscription over the portal of hell, 'Lasciate ogni speranza, voi ch' entrate.'

We have already seen that this objection is based upon entirely erroneous information as to the curability of the disease, but it is not the less likely to have a certain amount of weight.

2. There are again others who believe that those who endeavour to preserve the lives of these poor wretches, are doing a positive injury to the future of the human race.

This view, again, is based upon erroneous views as to the nature of the hereditary transmission of tubercle.

We have abundant reasons for believing that—by means of the destruction of the breeding-grounds of phthisis, and by improved hygienic measures, together with proper precautions against infection—the hereditary tendency to the disease would die out in a few generations, and that a healthy race would survive, free from tubercular taint, and endowed with the vivid intelligence and bright social attributes which are well known to be so common amongst those who now often succumb to the disease.

But to both these classes of objectors, as well as to others who have less excuse for parsimony in this regard, I would, in addition to meeting their arguments, adduce the powerful plea of self-interest.

Measures such as I have indicated would greatly diminish the danger of contracting the disease. Not only would some cases be entirely cured, but all of them would be restrained from spreading abroad the peculiar virus that might otherwise find a home in some susceptible system.

Moreover, such hospitals would be centres from which

would spread enlightened opinions respecting the proper treatment of the complaint, and they would be the means of preventing its further extension.

I do not hesitate to say that, at present, the hospital accommodation for cases of phthisis is most inadequate, and that in place of the half-dozen of such institutions outside the metropolis, there ought to be hundreds of them scattered about the country, in suitable localities and attached to all the chief centres of the population.

The map of the distribution of leper-houses in England, shows what large provision was made for leprosy at a time when the population was not much more than one-tenth of its present amount, and it is possible that advantage might be taken of some of the funds left for that purpose, to apply them to the treatment of a strictly analogous and closely allied form of disease.

I am glad to be able to quote Dr. Coats, of Glasgow, who is a high authority on this subject, in favour of the scheme now put forth. In his 'Lectures to Practitioners' (Longmans, Green & Co.), p. 211, he says: 'It may possibly in the future come to be a practical sanitary question whether consumptive patients should not, in some way, be isolated from the general community.'

'There can be no doubt that their presence in the general community, by continually breeding and giving out large quantities of tubercular bacilli, supplies one of the necessary elements in the propagation of all tubercular disease; it may indeed be a question whether, in the interests of the patients themselves, as well as of the community at large, it will not be advisable to undertake their treatment in isolated special hospitals. If consumptives were treated in sanatoriums, placed in dry, airy situations, and with efficient ventilation indoors, as well

as the opportunity of sufficient out-door exercise, then surely there would be a much larger proportion of cures than can be looked for under present circumstances.' He also adduces the example of leprosy and its treatment by isolation, and its success, as a case in point.

In conclusion, perhaps I may be allowed to say a few words as to the principles that should guide us in endeavouring to check the disease at its outset.

This point is indeed strictly germane to our subject of the prevention of phthisis, for if we could succeed in arresting its progress in a certain number of cases, we should by so much diminish the chance of its spreading to others.

I. May I point out the unsatisfactory results that have arisen so far from the various researches that have been made for the purpose of killing the bacillus *in situ* of late years ? The efforts of many physicians have been directed to this object, and the treatment, whether by inhalations, by sulphuretted hydrogen injections, or by germicide medicines, has had the one principle of endeavouring to destroy the bacillus after its entrance into the lungs. I was myself at one time beguiled into making attempts in this direction, but I have long been convinced of the extreme improbability of our ever reaching the organisms in their hiding-places in the lung.

A very little study of the pathology of the disease will convince most men that when the organism is hedged round, as it usually is, with the products of inflammatory action, with exudation-cells or fibroid material, and at a distance from vascular supply, it is almost hopeless to expect to reach it either through the air-passages or by the currents of the blood.

Let us have ' war to the bacillus,' by all means, but it must be war carried on outside the body and not

within it. When once it is entrenched behind its barrier of exudative material its individual existence, so far as we know, is safe against all our attacks.

II. What we have to do, after preventing reinforcements from being poured in from the outside, is to combat the irritation and inflammation caused by its presence by ordinary antiphlogistic measures, and then, by all the means in our power, so to strengthen the bodily forces as to enable them to deal with the invaders. If these receive no accession from without, and if they can be prevented from generating poisonous matter, more powerful for evil than themselves, they will generally succumb, either by starvation, or by being dried up, or got rid of by ordinary necrosis and suppuration.

To leave metaphor, I believe that our chief reliance must be placed upon means for re-invigorating the system—dietetic, hygienic, and medicinal.

III. At the same time, I would by no means discourage attempts to preserve adjacent structures and the rest of the body from attack. Certain of the inhalations most commonly used, such as carbolic acid, creasote, eucalyptus, oil of pine, menthol, iodine, &c., are probably of service in this direction, and some of them serve to control excessive secretion, and to render the mucous surfaces generally more healthy. It may be possible also that certain medicines, which have been proposed for this purpose, may prove, upon extended trial, to have the due preservative effect, and, hence, observations are needed upon the action of such drugs as tannin, creasote, iodoform, arsenic, salts of copper, &c., though I should myself give precedence to such as have been proved to increase the body-weight and to improve nutrition generally.

At the Manchester Hospital for Consumption, a series

of observations has been carried on for some years upon the action of different drugs, such as iodoform, the hypophosphites, guaiacol, creasote, tannin, and, latterly, upon the inhalation of pure ozonised oxygen; but it has been somewhat difficult to discriminate between the action of these substances and the general good influence of the hospital itself. Other workers in this field have also contributed evidence on this subject, but it is scarcely within the scope of this inquiry to attempt to sum up the results of these observations.

It may, however, be of some service if I remark again upon the inhalation of ozonised oxygen, which, so far as I know, has only been used as yet in the Manchester Hospital, so far with encouraging results.

It was first tried in consequence of the well-known beneficial action of pure mountain and sea air, and it was thought possible that some of this benefit might be due to the ozone contained in such air. After ascertaining that pure oxygen when ozonised up to 9 or 11 per cent. might be inhaled in considerable quantities without exciting inflammatory action, it was administered regularly to fifteen patients in all stages of the complaint, and the results were very marked in procuring improvement in general health—better appetite, sounder sleep, freedom from fever, and consequent gain in weight.[1]

And yet it certainly had no obvious germicidal action in many of the cases; though the amount of expectoration was diminished, there was but little difference to be noted in the number of bacilli on the microscopic slides.

IV. So far as I know, all the attempts that have been made, hitherto chiefly in France,[2] to discover an antibacil-

[1] See Report in *Med. Chron.*, May 1889.

[2] See especially papers by Gosselin, Jeannel, Laulanié, and Martin in Verneuil's series of *Etudes Expérimentales sur la Tuberculose.*

lary vaccine, with which to render the tissues antagonistic to the bacillus, have signally failed; and although I would not object to further researches in this direction, it appears to be hardly likely that they will be successful against a complaint in which one attack confers no immunity against a second.

V. Hitherto the best results that have been obtained in the cure of consumption have been due to abundant supplies of fresh air and light and good food, with a large abundance of fat in an easily assimilated condition, and to medicines that have assisted the general nutrition of the body.

VI. But if the question were to be addressed to medical men by the Collective Investigation Committee, as to what simple measure has done most towards the cure of consumption, I believe the answer would be almost unanimous, in favour of change of residence. In many instances, no doubt, the improvement would be ascribed to change of climate, and sometimes Egypt or Algiers, sometimes Davos, or Canada, or the Riviera, would receive the praise. Much good must undoubtedly arise from the favourable conditions to be found in these places, but I venture to ascribe a large proportion of the cures to the simple fact of removal from an infected area, to places where the air is free from active virus—and hence I am at one with those who believe that healthy homes for consumptives may be found in this country.

I have made these few remarks in the belief that your College would prefer me to state plainly the convictions that have grown upon me in the course of a practice during which diseases of the lungs have long been especially the object of my study.

But all the conclusions now stated are obviously sub-

ject to revision in the light of evidence that may be forthcoming as the result of further research.[1]

The subject is still *sub judice*, but when the final judgment comes to be pronounced I only hope that there will remain sufficient ground for encouragement to persevere in the task of meeting and ultimately annihilating this terrible disease.

From a review of the course of this complaint in the past, and of its affinities with other preventible disorders —from our knowledge of its pathology, and of the influences most favourable to its spread—from our experience of its steady diminution within the last thirty years, and from the brief *résumé* of the measures that may be taken to arrest its course—from all these points we may surely learn to regard it as a preventible disease, and may look forward to its further diminution, if not to its ultimate extinction as a cause of death.

[1] I would call attention to Dr. Vincent Harris's recent paper 'On the Antiseptic Treatment of Phthisis' (*St. Barth.'s Hosp. Reports*, xxv. 49); but I may observe that equally good results have been obtained in hospitals, apart from the use of antiseptic inhalations.

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